Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=YES; OP=ADJ			
L10	L3 and g protein coupled receptor	27	L10
L9	L6 and g protein coupled receptor	16	L9
L8	L7 and g protein coupled receptor	15	L8
L7	matsui-hideki.in.	377	L7
L6	terao-yasuko.in.	28	L6
L5	terao-yasuko-.in.	28	L5
L4	wananaba-takuya.in.	0	L4
L3	L1 with mas	27	L3
L2	L1 and mas	1562	L2
L1	g protein coupled receptor	5072	L1

END OF SEARCH HISTORY

WEST

Help Logout Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Term	Documents
G	3111622
GS	16913
PROTEIN	294868
PROTEINS	185306
COUPLED	1424845
COUPLEDS	5
RECEPTOR	135051
RECEPTORS	78613
(3 AND (((G ADJ PROTEIN) ADJ COUPLED) ADJ RECEPTOR)).USPT,PGPB,JPAB,EPAB,DWPI,TDBD.	27
(L3 AND G PROTEIN COUPLED RECEPTOR).USPT,PGPB,JPAB,EPAB,DWPI,TDBD.	27

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L10

Refine Search

Recall Text Clear

Search History

DATE: Friday, October 17, 2003 Printable Copy Create Case

FILE 'MEDLINE'
FILE 'JAPIO'
FILE 'BIOSIS'
FILE 'SCISEARCH'
FILE 'WPIDS'
FILE 'CAPLUS'
FILE 'EMBASE'
=> s g protein coupled receptor
5 FILES SEARCHED...
L1 43587 G PROTEIN COUPLED RECEPTOR

=> s l1 and mas
L2 182 L1 AND MAS

=> dup rem l2
PROCESSING IS APPROXIMATELY 87% COMPLETE FOR L2
PROCESSING COMPLETED FOR L2
L3 76 DUP REM L2 (106 DUPLICATES REMOVED)

=> d l3 ibib abs 1-76

L3 ANSWER 1 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 1
ACCESSION NUMBER: 2003-541408 [51] WPIDS
DOC. NO. CPI: C2003-146823
TITLE: Preparation of formulations of the peptide angiotensin-(1-7) and/or its derivatives, useful for treating e.g. tumor, involves encapsulation of the peptide and/or its derivatives in liposomes, cyclodextrins and polymers.
DERWENT CLASS: A96 B04
INVENTOR(S): DOS SANTOS, R A S; FREZAD, F J G; MILLAN, R D S; NADU, A P
PATENT ASSIGNEE(S): (UYMI-N) UNIV FEDERAL MINAS GERAIS UFMG
COUNTRY COUNT: 100
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003039434	A2	20030515	(200351)*	EN	27

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU
MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT
RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM
ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003039434	A2	WO 2002-BR156	20021105

PRIORITY APPLN. INFO: BR 2001-5509 20011105
AN 2003-541408 [51] WPIDS
AB WO2003039434 A UPAB: 20030808
NOVELTY - Preparation of formulations of the peptide angiotensin-(1-7) and/or its analogs and derivatives, including Sar1-angiotensin(1-7), involves use of the cyclodextrins, their derivatives, liposomes, polymers and/or their derivatives.
ACTIVITY - Hypotensive; Cardiovascular; CNS; Vulnerary; Cytostatic; Antidiabetic; Gynecological; Nephrotropic; Gastrointestinal; Angiogenesis inhibition; Angiogenesis stimulator; Endocrine.
The hypotensive activity was evaluated by using the formulation of the angiotensin-(1-7) encapsulated in liposomes comprising distearoyl-phosphatidylcholine, cholesterol and distearoyl-phosphatidylethabolamine-polyethylene glycol (2000) at a ratio of 5:4:0.3 (test). The formulation was microinjected (35 ng of ang-(1-7) in 200 nl) in the rostroventrolateral medulla of Wistar rats. Empty liposomes (control) were also similarly microinjected at the same lipid dose. The mean arterial blood pressure (MAP) was determined 12 days after microinjection. The microinjection of the angiotensin peptide produced a significant pressure effect during daytime which sustained for 5 days. The daytime MAP was higher (114 plus or minus 4 mm Hg) on day 3 than that in control (100 plus or minus 3 mm Hg).
MECHANISM OF ACTION - ***MAS***. receptor agonist and antagonist.
USE - For preparing formulations of the peptide angiotensin-(1-7) (e.g. (A779) D-Ala7-angiotensin-(1-7) and D-Pro7-angiotensin-(1-7)), and/or its analogs and derivatives, including Sar1-angiotensin(1-7), which is used in the study and treatment of arterial hypertension, other cardiovascular diseases and its complications, wounds, burns, erythema, tumor, diabetes mellitus, sperm mobility, renal disease (e.g. nephropathy), gastrointestinal and gynecological disorders, angiogenesis, angioplasty, alopecia, blood and cerebral diseases in warm blooded animals. For the identification of a ligand receptor and a similar interaction between the ***G*** - ***protein*** - ***coupled*** ***receptor*** , ***MAS*** , and angiotensin-(1-7) or its analogs and derivatives in optionally encapsulated form (all claimed).
ADVANTAGE - The formulation prepared by the process provides the increase of the duration through sustained release of the drug and improves efficacy of the biological effects. The formulation decreases the degradation of the peptide in the treatment of gastrointestinal, hence increases bio-dispensability of the peptide in the biological system. The formulation facilitates identification of interaction of ***MAS*** with the peptide.
Dwg.0/0

L3 ANSWER 2 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:532691 CAPLUS
DOCUMENT NUMBER: 139:95435
TITLE: Modified receptors on cell membranes for the discovery of therapeutic ligands
INVENTOR(S): Schwartz, Thue W.; Martini, Lene; Heydorn, Arne; Jorgensen, Rasmus
PATENT ASSIGNEE(S): 7TM Pharma A/S, Den.
SOURCE: PCT Int. Appl., 122 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003055914	A2	20030710	WO 2002-DK900	20021220

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: DK 2001-1944 A 20011221
DK 2002-113 A 20020122
DK 2002-1043 A 20020703
US 2002-394122P P 20020703

AB A drug discovery method is provided for selecting a compd. selected from the group consisting of a small org. substance, a biopharmaceutical, or an antibody or part thereof. The method comprises the steps of (i) expressing one or more receptors on a cell membrane, such as, e.g., an exterior cell surface of a cell, (ii) contacting one or more expressed receptors with a test compd. or a selection of test compds. (libraries), and (iii) selecting one or more compds. based on its ability to bind one or more receptors. The step of expressing the one or more receptors comprises capturing one or more receptors on the exterior cell surface in a conformation that predominantly enables binding or interaction with a ligand, and the conformation that predominantly enables binding or interaction with a ligand is provided by modification of one or more receptors by a method comprising at least one of the following: (a) fusion with any protein which keeps the receptor in the desired conformation such as, e.g. an arrestin, a modified arrestin, a G-protein or a modified G-protein, (b) site-directed mutagenesis, and (c) deletion. The receptors may be captured on the exterior cell surface by at least one of the following: (d) interaction of the receptor with a scaffolding protein, optionally, with a scaffolding protein network and (e) means for blocking receptor internalization, e.g. by co-expression of a mutated dynamin or a modified arrestin or by use of chems. such as, e.g., sucrose and/or Tris. Thus, by coexpressing of either the wild-type receptor or by modifying the receptor by engineering for example a recognition motif for a strong binder into its structure (for example, a PDZ recognition motif at its C-terminal end), and coexpression of this with a scaffolding protein such as PSD-95 or a modified scaffolding protein which interacts with the cytoskeleton at the cell surface or is made to be closely assocd. with the membrane through a lipid anchor, a high level of surface expression can be ensured, which will benefit its use in the drug discovery process. As a result of the strong tendency of the scaffolding proteins to interact with each other, just the cotransfection with one or more appropriate scaffolding proteins or modified scaffolding protein may also lead to the formation of patches with high local concns of the receptor or modified receptor, which will be highly beneficial in the drug discovery process where they are used initially to select binding mols. The method is exemplified by expression of the NK1 receptor in an agonist high-affinity binding form at the surface of transfected cells through fusion with arrestin or the N-terminal fragment of arrestin.

L3 ANSWER 3 OF 76 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2003391800 IN-PROCESS
DOCUMENT NUMBER: 22810130 PubMed ID: 12909716
TITLE: Atypical expansion in mice of the sensory neuron-specific Mrg ***G*** ***protein*** - ***coupled*** ***receptor*** family.
AUTHOR: Zylka Mark J; Dong Xinzhong; Southwell Amber L; Anderson David J
CORPORATE SOURCE: Division of Biology, 216-76, and Howard Hughes Medical Institute, California Institute of Technology, Pasadena, CA 91125.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2003 Aug 19) 100 (17) 10043-8.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
OTHER SOURCE: GENBANK-AF518238; GENBANK-AF518239; GENBANK-AF518240; GENBANK-AF518241; GENBANK-AF518242; GENBANK-AF518243; GENBANK-AF518244; GENBANK-AF518245; GENBANK-AF518246; GENBANK-AF518247; GENBANK-AF518248; GENBANK-AF518249; GENBANK-AY196926; GENBANK-AY196927; GENBANK-AY196928; GENBANK-AY266420
ENTRY DATE: Entered STN: 20030821
Last Updated on STN: 20030903
AB The ***Mas*** -related genes (Mrgs) comprise a family of >50 ***G*** ***protein*** - ***coupled*** ***receptors*** (GPCRs), many of which are expressed in specific subsets of nociceptive sensory neurons in mice. In contrast, humans contain a related but nonorthologous family of genes, called MrgXs or sensory neuron-specific receptors, of which many fewer appear to be expressed in sensory neurons. To determine whether the diversity of murine Mrgs is generic to rodents or is an atypical feature of mice, we characterized MrgA, MrgB, MrgC, and MrgD subfamilies in rat and gerbil. Surprisingly, although mice have approximately 22 MrgA and approximately 14 MrgC genes, rats and gerbils have just a single MrgA and MrgC gene. This murine-specific expansion likely reflects recent retrotransposon-mediated unequal crossover events. The expression of Mrgs in rat sensory ganglia suggests that the extensive cellular diversity in mice can be simplified to a core subset of approximately four different genes (MrgA, MrgB, MrgC, and MrgD), defining a similar number of neuronal subpopulations. Our results suggest more generally that mouse-human genomic comparisons may sometimes reveal differences atypical of rodents.

L3 ANSWER 4 OF 76 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2003322017 MEDLINE
DOCUMENT NUMBER: 22735888 PubMed ID: 12829792
TITLE: Angiotensin-(1-7) is an endogenous ligand for the ***G*** ***protein*** - ***coupled*** ***receptor*** ***Mas*** .
AUTHOR: Santos Robson A S; Simoes e Silva Ana C; Maric Christine; Silva Denise M R; Machado Raquel Pillar; de Buhr Insa; Heringer-Walther Silvia; Pinheiro Sergio Veloso B; Lopes Myriam Teresa; Bader Michael; Mendes Elizabeth P; Lemos Virgina Soares; Campagnole-Santos Maria Jose; Schultheiss Heinz-Peter; Speth Robert; Walther Thomas
CORPORATE SOURCE: Department of Physiology and Biophysics, Federal University of Minas Gerais, Belo Horizonte, 31270, Minas Gerais, Brazil.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2003 Jul 8) 100 (14) 8258-63.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200309
ENTRY DATE: Entered STN: 20030710
Last Updated on STN: 20030905
Entered Medline: 20030904
AB The renin-angiotensin system plays a critical role in blood pressure control and body fluid and electrolyte homeostasis. Besides angiotensin (Ang) II, other Ang peptides, such as Ang III [Ang-(2-8)], Ang IV [Ang-(3-8)], and Ang-(1-7) may also have important biological activities.
Ang-(1-7) has become an angiotensin of interest in the past few years, because its cardiovascular and baroreflex actions counteract those of Ang II. Unique angiotensin-binding sites specific for this heptapeptide and studies with a selective Ang-(1-7) antagonist indicated the existence of

a
distinct Ang-(1-7) receptor. We demonstrate that genetic deletion of the
G ***protein*** - ***coupled*** ***receptor*** encoded
by the ***Mas*** protooncogene abolishes the binding of Ang-(1-7) to
mouse kidneys. Accordingly, ***Mas*** -deficient mice completely lack
the antidiuretic action of Ang-(1-7) after an acute water load. Ang-(1-7)
binds to ***Mas*** -transfected cells and elicits arachidonic acid release. Furthermore, ***Mas*** -deficient aortas lose their
Ang-(1-7)-induced relaxation response. Collectively, these findings identify ***Mas*** as a functional receptor for Ang-(1-7) and provide
a clear molecular basis for the physiological actions of this biologically active peptide.

L3 ANSWER 5 OF 76 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2003247863 MEDLINE
DOCUMENT NUMBER: 22656136 PubMed ID: 12769986
TITLE: Myotropic effect of helicokinins, tachykinin-related peptides and Manduca sexta allatotropin on the gut of Heliothis virescens (Lepidoptera: Noctuidae).
AUTHOR: Oeh U; Antonicek H; Nauen R
CORPORATE SOURCE: Bayer AG, Bayer CropScience, Global Biology Insecticides,
Building 6220, Alfred-Nobel-Strasse 50, 40789 Monheim, Germany.
SOURCE: JOURNAL OF INSECT PHYSIOLOGY, (2003 Apr) 49 (4) 323-37.
Journal code: 2985080R. ISSN: 0022-1910.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200308
ENTRY DATE: Entered STN: 20030529
Last Updated on STN: 20030809
Entered Medline: 20030808
AB Different insect neuropeptides (helicokinins, tachykinin-related and allatoregulating peptides) were investigated with regard to their myostimulatory effects using whole-gut preparations isolated from fifth instar Heliothis virescens larvae. The experiments demonstrated that representatives of all three peptide families are able to induce and amplify gut contractions in this species in a dose-dependent manner. Structure-activity studies (alanine scan, D-amino acid scan and truncated
analogues) with the helicokinin Hez-K1 supported the finding, that the core sequence for biological activity of kinins is the amidated C-terminal pentapeptide (FSPWG-amide). Similar investigations with insect tachykinin
isolated from Leucophaea madera (Lem-TRP1) revealed that the minimum
sequence evoking a physiological gut response in H. virescens is the amidated hexapeptide (GFLGVR-amide), which represents the conserved amino
acid sequence for Leucophaea TRPs in general. The peptide concentration
causing a half-maximal gut contraction (EC(50)) for Lem-TRP1 was about 26
nM. Although the potency of Lem-TRP1 was 9-fold lower compared with
Hez-KI (EC(50): 3 nM), the maximal tension of the gut obtained with Lem-TRP1 was 1.7-fold higher compared with Hez-KI. The EC(50) of Manduca
sexta allatotropin (***Mas*** -AT; 79 nM) was of lowest potency among
all three peptides tested. In a pharmacological study, co-incubation experiments with Lem-TRP1, Hez-KI or ***Mas*** -AT and compounds
interfering with signal transduction pathways were employed to investigate
the mode of action of the myotropic effects of these peptides. Cadmium
and the protein kinase C (PKC) inhibitor tamoxifen attenuated the contractile effects of all three peptides tested. The data suggest that in the gut muscle of H. virescens the myotropic peptides bind to ***G***
- ***protein*** - ***coupled*** ***receptors*** that cause
contraction by promoting the entry of extracellular calcium mediated by a
PKC involved pathway.

L3 ANSWER 6 OF 76 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:307059 BIOSIS
DOCUMENT NUMBER: PREV200300307059
TITLE: The ***G*** ***protein*** - ***coupled*** ***receptor*** ***Mas*** is a physiological antagonist of the angiotensin II AT1 receptor.
AUTHOR(S): Walther, Th. (1); Milligan, G.; Christopoulos, A.; Sanchez-Ferrer, C.; Heringer-Walther, S. (1); Sexton, P.; Schultheiss, H.-P. (1); Kostenis, E.
CORPORATE SOURCE: (1) Dept. of Cardiology, University Hospital Benjamin
Franklin (UKBF), Freie Universitaet Berlin, 12200, Berlin, Germany Germany
SOURCE: Naunyn-Schmiedeberg's Archives of Pharmacology, (March
2003, 2003) Vol. 367, No. Supplement 1, pp. R19. print.
Meeting Info.: 44th Spring Meeting of the Deutsche Gesellschaft fuer Experimentelle und Klinische Pharmakologie und Toxikologie and the 20th Meeting of the Gesellschaft fuer Umwelt-Mutationsforschung Mainz, Germany
March 17-20, 2003
ISSN: 0028-1298.
DOCUMENT TYPE: Conference
LANGUAGE: English

L3 ANSWER 7 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 2003:766670 SCISEARCH
THE GENUINE ARTICLE: 670XN
TITLE: The ***G*** ***protein*** - ***coupled*** ***receptor*** ***Mas*** is a physiological antagonist of the angiotensin II AT1 receptor
AUTHOR: Walther T (Reprint); Milligan G; Christopoulos A; Sanchez-Ferrer C; Heringer-Walther S; Sexton P; Schultheiss H P; Kostenis E
CORPORATE SOURCE: Free Univ Berlin, Univ Hosp Benjamin Franklin, Dept
Cardiol, D-12200 Berlin, Germany; Univ Glasgow, Inst Biomed & Life Sci, Glasgow, Lanark, Scotland; Univ Melbourne, Dept Pharmacol, Melbourne, Vic, Australia; Univ
Autonoma Madrid, Fac Med, Dept Pharmacol, Madrid, Spain;
Univ Melbourne, Howard Florey Inst Expt Physiol & Med, Melbourne, Vic, Australia; Univ Copenhagen, Inst Pharmacol, Copenhagen, Denmark
COUNTRY OF AUTHOR: Germany; Scotland; Australia; Spain; Denmark
SOURCE: NAUNYN-SCHMIEDEBERGS ARCHIVES OF PHARMACOLOGY, (MAR 2003)
Vol. 367, Supp. [1], pp. R19-R19. MA 63.
Publisher: SPRINGER-VERLAG, 175 FIFTH AVE, NEW YORK, NY
10010 USA.
ISSN: 0028-1298.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0

L3 ANSWER 8 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2003-018588 [01] WPIDS
CROSS REFERENCE: 2001-541358 [60]; 2003-532533 [50]
DOC. NO. NON-CPI: N2003-014435
DOC. NO. CPI: C2003-004377
TITLE: Validating the therapeutic or pharmacological potential of target molecules e.g. receptors by using a genetically modified animal which expresses a silent metal-ion site in a potential drug target.
DERWENT CLASS: A96 B04 B05 D16 S03
INVENTOR(S): LANGE, B H; RIST, O; SCHWARTZ, T W
PATENT ASSIGNEE(S): (SEVE-N) 7TM PHARMA AS
COUNTRY COUNT: 99
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002054077	A2	20020711	(200301)*	EN	78

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT
RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002054077	A2	WO 2001-DK867	20011221

PRIORITY APPLN. INFO: US 2001-280237P 20010330; WO 2000-EP13389
20001229; DK 2001-536 20010330
AN 2003-018588 [01] WPIDS
CR 2001-541358 [60]; 2003-532533 [50]
AB WO 200254077 A UPAB: 20030805
NOVELTY - Target validation process for testing or validating physiological importance, therapeutic of biological target molecule (TM),
involves introducing silent metal ion site (SMIS) in TM to obtain an engineered TM, testing a compound for its ability to bind to the SMIS in
the TM, testing the compound in a genetically modified test animal with the engineered TM and monitoring certain parameters of the animal.
DETAILED DESCRIPTION - A target validation process for testing or
validating physiological importance, therapeutic of a biological target molecule (TM), involves introducing a silent metal ion site (SMIS) in TM
to obtain an engineered TM, testing a compound for its ability to bind to
the SMIS in the TM, testing the compound in a genetically modified test
animal with the engineered TM and monitoring certain parameters of the
animal. The method comprises:
(i) introduction of a silent metal ion site in TM to obtain a silent metal ion engineered TM;
in vitro testing of a test compound for its ability to bind to the introduced silent metal ion site in the silent metal ion engineered TM;
(ii) optionally, chemically optimizing the test compound and/or the biological TM to create secondary interaction(s) with chemical groups in
the vicinity of the metal ion site in the silent metal ion engineered TM;
(iii) optionally, repeating ii) and iii) and to obtain a suitable binding affinity in the in vitro test;
(iv) optionally, chemically optimizing the test compound to improve the pharmacokinetic and/or biopharmaceutical properties of the test compound;
(v) preparing a genetically modified test animal containing the silent metal ion site engineered TM; and
in vivo testing of the optionally optimized test compound in the genetically modified test animal, and monitoring the biochemical, physiological and/or behavior parameters of the test animal.
USE - The method is useful for target validation process for testing or validating physiological importance and/or therapeutic of a biological TM such as proteins, polypeptides, oligopeptides, nucleic acids, carbohydrates, nucleoproteins, glycoproteins, glycolipids, lipoproteins and their derivatives, membrane receptors, signal transduction proteins, scaffolding proteins, nuclear receptors, steroid receptors, intracellular receptors, transcription factors, enzymes, allosteric enzyme regulator proteins, growth factors, hormones, neuropeptides or immunoglobulins.
Dwg.0/5

L3 ANSWER 9 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2002-154854 [20] WPIDS
CROSS REFERENCE: 2003-265771 [26]
DOC. NO. NON-CPI: N2002-117729
DOC. NO. CPI: C2002-048442
TITLE: Novel non-human transgenic animal, preferably transgenic
mice comprising disruptions in target ***G*** ***protein*** - ***coupled*** ***receptor*** gene, useful for identifying an agent that modulates expression or function of target gene.
DERWENT CLASS: B04 D16 P14
INVENTOR(S): ALLEN, K D; BRENNAN, T J
PATENT ASSIGNEE(S): (DELT-N) DELTAGEN INC; (ALLE-I) ALLEN K D; (BREN-I)
BRENNAN T J
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002003789	A2	20020117	(200220)*	EN	93

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ
LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD
SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001071902 A 20020121 (200234)
US 2002082232 A1 20020627 (200245)
US 2002088018 A1 20020704 (200247)
US 2002148001 A1 20021010 (200269)
US 2002162132 A1 20021031 (200274)
US 2003033624 A1 20030213 (200314)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002003789	A2	WO 2001-US21498	20010706
AU 2001071902	A	AU 2001-71902	20010706
US 2002082232	A1 Provisional	US 2000-216260P	20000706
	Provisional	US 2000-221474P	20000727
		US 2001-900497	20010706
US 2002088018	A1 Provisional	US 2000-216250P	20000706
	Provisional	US 2000-223625P	20000807
		US 2001-900472	20010706
US 2002148001	A1 Provisional	US 2000-216475P	20000706
	Provisional	US 2000-221473P	20000727
		US 2001-900700	20010706
US 2002162132	A1 Provisional	US 2000-216253P	20000706
	Provisional	US 2000-219403P	20000719
	Provisional	US 2000-251815P	20001206
	Provisional	US 2001-262137P	20010116
		US 2001-900699	20010706
US 2003033624	A1 Provisional	US 2000-216254P	20000706
	Provisional	US 2000-221497P	20000727
	Provisional	US 2001-280264P	20010329
		US 2001-900519	20010706

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001071902	A Based on	WO 2002003789

PRIORITY APPLN. INFO: US 2001-300929P 20010626; US 2000-216108P
20000706; US 2000-216250P 20000706; US 2000-216252P 20000706; US 2000-216253P 20000706; US 2000-216254P 20000706; US 2000-216259P 20000706; US 2000-216260P 20000706; US 2000-216271P 20000706; US 2000-216473P 20000706; US 2000-216475P 20000706; US 2000-219403P 20000719; US 2000-221473P 20000727; US 2000-221474P 20000727; US 2000-221484P 20000727; US 2000-221490P 20000727; US 2000-221497P 20000727; US 2000-223625P 20000807; US 2000-251815P 20001206; US 2001-262137P

20010116; US 2001-262138P 20010116; US 2001-280264P 20010329; US 2001-900497 20010706; US 2001-900472 20010706; US 2001-900700 20010706; US 2001-900699 20010706; US 2001-900519 20010706

AN 2002-154854 [20] WPIDS
CR 2003-265771 [26]
AB WO 200203789 A UPAB: 20030428
NOVELTY - Non-human transgenic animal (I) with targeted ***G*** ***protein*** - ***coupled*** ***receptor*** gene disruption in DEZ receptor gene, CB2R gene, adrenomedullin receptor gene, corticotropin-releasing factor receptor (CRFR2) gene, N-formylpeptide receptor-like 3 (FPRL3) gene, neuropeptide Y receptor gene (NPY4-R, NPY6-R), kappa-3 opiate receptor (KOR3) gene, metabotropic glutamate receptor (mGluR8) gene, ***MAS*** receptor gene, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) a cell (II) derived from (I);
(2) a targeting construct (III) comprising first and second polynucleotide sequences homologous to the target gene, and a selectable marker;
(3) a cell (IV) comprising a disruption in the target gene;
(4) production of (III);
(5) producing (I), preferably a transgenic mouse, by introducing (III) into a cell, introducing the cell into a blastocyst, implanting the resulting blastocyst into a pseudopregnant mouse, where the pseudopregnant mouse gives birth to a chimeric mouse, and breeding the chimeric mouse;
(6) an agent (V) that modulates the expression or function of the target gene, or a phenotype associated with (I), identified using (I);
(7) phenotypic data (VI) associated with (I), where the data is in a data base; and
(8) an agonist or antagonist (VII) of DEZ receptor, an adrenomedullin receptor, CRFR2, NPY6-R, mGluR8 or ***MAS*** receptor.
ACTIVITY - None given.
MECHANISM OF ACTION - Modulator of expression or function of the target gene; modulator of phenotype associated with (I) (claimed). No supporting data is given.
USE - (I) or (IV) is useful for identifying an agent that modulates the expression or function of the target gene, by providing (I) or (IV), administering an agent to (I) or contacting (IV) with an agent, and determining whether the expression or function of the target is modulated.
(I) is useful for identifying an agent that modulates a phenotype associated with (I), by administering an agent to (I), and determining whether the agent modulates the phenotype. The phenotypic data associated with the mouse is in a database (claimed). (I) is useful for testing the efficacy of proposed genetic and pharmacological therapies for human genetic diseases, such as neurological, neuropsychological or psychotic illnesses. (I) or (IV) is useful as models for diseases, disorders or conditions associated with phenotypes relating to a disruption in a target, and to identify drugs, pharmaceuticals, therapies and interventions which may be effective in treating a disease or other phenotypic characteristics of the animal. (V) is useful as a therapeutic for treating conditions associated with a disruption of the target gene.
Dwg.0/30

L3 ANSWER 10 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:676195 CAPLUS
DOCUMENT NUMBER: 137:227713
TITLE: Human cDNA sequences and their encoded proteins and diagnostic and therapeutic uses
INVENTOR(S): Tchernev, Velizar T.; Spytek, Kimberly A.; Zerhusen, Bryan D.; Patturajan, Meera; Shimkets, Richard A.; Li, Li; Gangolli, Esha A.; Padigaru, Muralidhara; Anderson, David W.; Rastelli, Luca; Miller, Charles E.; Gerlach, Valerie L.; Taupier, Raymond J., Jr.; Gusev, Vladimir Y.; Colman, Steven D.; Wolenc, Adam R.; Pena, Carol E. A.; Furtak, Katarzyna; Grosse, William M.; Alsobrook, John P., II; Lepley, Denise M.; Rieger, Daniel K.; Burgess, Catherine E.
PATENT ASSIGNEE(S): Curagen Corporation, USA
SOURCE: PCT Int. Appl., 1498 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 60
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002068649	A2	20020906	WO 2002-US2785	20020131

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-265395P P 20010131
US 2001-265412P P 20010131
US 2001-265514P P 20010131
US 2001-265517P P 20010131
US 2001-266406P P 20010202
US 2001-266767P P 20010205
US 2001-266975P P 20010207
US 2001-267057P P 20010207
US 2001-267459P P 20010208
US 2001-267823P P 20010209
US 2001-268974P P 20010215
US 2001-271664P P 20010226
US 2001-271839P P 20010227
US 2001-271855P P 20010227
US 2001-272788P P 20010302
US 2001-273046P P 20010302
US 2001-275925P P 20010314
US 2001-275947P P 20010314
US 2001-275950P P 20010314
US 2001-275989P P 20010314

AB Disclosed herein are 162 cDNA sequences that encode novel human polypeptides that are members of the various protein families. Also disclosed are polypeptides encoded by these nucleic acid sequences, and antibodies, which immunospecifically-bind to the polypeptide, as well as derivs., variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, treatment, and prevention of disorders involving any one of these novel human nucleic acids and proteins.

L3 ANSWER 11 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:332342 CAPLUS
DOCUMENT NUMBER: 136:351432
TITLE: Protein, gene and cDNA sequences of a novel human ***G*** ***protein*** - ***coupled*** ***receptor*** sequence homolog and diagnostic and therapeutic uses thereof
INVENTOR(S): Wei, Ming-Hui; Zhao, Qi; Woodage, Trevor; Di Francesco, Valentina; Beasley, Ellen M.
PATENT ASSIGNEE(S): PE Corporation (NY), USA
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002034914	A1	20020502	WO 2001-US31592	20011010

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002013082	A5	20020506	AU 2002-13082	20011010
EP 1330522	A1	20030730	EP 2001-981441	20011010

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRIORITY APPLN. INFO.: US 2000-695045 A 20001025
US 2001-867570 A 20010531
WO 2001-US31592 W 20011010

AB The invention provides protein, cDNA and genomic sequences for a novel human protein, which shares sequence homol. to a known ***G*** ***protein*** - ***coupled*** ***receptor*** , and is related to ***Mas*** -related ***G*** ***protein*** - ***coupled*** ***receptor*** subfamily. The gene is expressed in human erythroleukemia cells and testis. The novel ***Mas*** -related ***G*** ***protein*** - ***coupled*** ***receptor*** gene has been mapped to human chromosome 3. Thus, the present invention specifically provides isolated peptide and nucleic acid mols., methods of identifying orthologs and paralogs of the ***G*** ***protein*** - ***coupled*** ***receptor*** peptides, methods of identifying modulators of the ***G*** ***protein*** - ***coupled*** ***receptor*** peptides, and methods of diagnosis and treatment of diseases assocd. with the ***G*** ***protein*** - ***coupled*** ***receptor*** .

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 12 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:409179 CAPLUS
DOCUMENT NUMBER: 137:1562
TITLE: Protein, DNA and cDNA sequences of human ***G*** - ***protein*** ***coupled*** ***receptor*** sequence homolog, and biological uses thereof, including use in drug screening
INVENTOR(S): Wei, Ming-hui; Shao, Wei; Zhao, Qi; Di Francesco, Valentina; Beasley, Ellen M.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 36 pp., Cont.-in-part of U.S. Ser. No. 634,881.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002064822	A1	20020530	US 2001-816087	20010326
WO 2002077015	A1	20021003	WO 2002-US9083	20020326

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003059891	A1	20030327	US 2002-266643	20021009

PRIORITY APPLN. INFO.: US 2000-634881 A2 20000807
US 2001-816087 A 20010326

AB The invention provides an isolated human protein, that based on sequence homol. and protein motifs, was referred to as a ***G*** - ***protein*** ***coupled*** ***receptor*** (GPCR). The invention also provides nucleic acid mols. (cDNA and DNA) encoding the human GPCR sequence homolog, and use of said nucleic acid mols. in construction of an expression vector for use in recombinant protein prodn.
The invention further provides an antibody specific for the human GPCR sequence homolog, and methods for detecting said GPCR protein, and said nucleic acid mols. Still further, the invention provides for the use of said human GPCR sequence homolog in assays for detecting agents that bind to GPCR and act as modulators, wherein said modulators can be used for treating a disease or disorder mediated by human protease. Finally, the invention provides the cDNA and genomic sequences, as well as amino acid sequence, of the human GPCR sequence homolog. The invention related that the human GPCR sequence homolog is related to the ***MAS*** proto-oncogene receptor subfamily. The invention also presented a list of tissues where GPCR sequence homolog expression was found, which included human brain, leukocytes, kidney, testis, bone marrow, small intestine and placenta. Further, the invention provided information on single polymorphisms (SNP) found in the human GPCR sequence homolog gene, and information on putative protein motifs of the human GPCR sequence homolog.

L3 ANSWER 13 OF 76 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2002669569 MEDLINE
DOCUMENT NUMBER: 22317401 PubMed ID: 12397184
TITLE: Orphan ***G*** ***protein*** - ***coupled*** ***receptors*** MrgA1 and MrgC11 are distinctively activated by RF-amide-related peptides through the Galpha q/11 pathway.
AUTHOR: Han Sang-Kyou; Dong Xinzhong; Hwang Jong-Ik; Zylka Mark J; Anderson David J; Simon Melvin I
CORPORATE SOURCE: Division of Biology 147-75, California Institute of Technology, Pasadena 91125, USA.
CONTRACT NUMBER: GM-34236 (NIGMS)
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2002 Nov 12) 99 (23) 14740-5.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200301
ENTRY DATE: Entered STN: 20021114
Last Updated on STN: 20030116
Entered Medline: 20030115

AB MrgA1 and MrgC11 belong to a recently identified family of orphan ***G*** - ***protein*** ***coupled*** ***receptors*** , called mrgs (***mas*** -related genes). They are only expressed in a specific

subset of sensory neurons that are known to detect painful stimuli. However, the precise physiological function of Mrg receptors and their underlying mechanisms of signal transduction are not known. We therefore have screened a series of neuropeptides against human embryonic kidney (HEK) 293 cells that stably express either MrgA1 or MrgC11 to identify ligands and/or agonists. MrgA1- or MrgC11-specific agonists stimulated dose-dependent increases in intracellular free Ca(2+) in a pertussis toxin-insensitive manner, but failed to alter basal or *forskolin-stimulated levels of intracellular cAMP. Furthermore, studies* using embryonic fibroblasts derived from various Galpha protein knockout mice demonstrated that both the MrgA1 and MrgC11 receptors are coupled to the Galpha(q/11) signaling pathway. Screening of neuropeptides identified surrogate agonists, most of these peptides included a common C-terminal -RF(Y)G or -RF(Y) amide motif. Structure-function studies suggest that endogenous ligands of Mrg receptors are likely to be RF(Y)G and/or RF(Y) amide-related peptides and that postprocessing of these peptides may serve to determine Mrg receptor-ligand specificity. The differences in ligand specificity also suggest functional diversity amongst the Mrg receptors.

L3 ANSWER 14 OF 76 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 2002365639 MEDLINE
DOCUMENT NUMBER: 22103682 PubMed ID: 12093898
TITLE: (1)H and (13)C ***MAS*** NMR evidence for pronounced ligand-protein interactions involving the ionone ring of the retinylidene chromophore in rhodopsin.
AUTHOR: Creemers Alain F L; Kiihne Suzanne; Bovee-Geurts Petra H M; DeGrip Willem J; Lugtenburg Johan; de Groot Huub J M
CORPORATE SOURCE: Leiden Institute of Chemistry, Leiden University, P.O. Box 9502, 2300 RA, Leiden, The Netherlands.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2002 Jul 9) 99 (14) 9101-6.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200208
ENTRY DATE: Entered STN: 20020712
Last Updated on STN: 20030105
Entered Medline: 20020808
AB Rhodopsin is a member of the superfamily of ***G*** - ***protein*** - ***coupled*** ***receptors*** . This seven alpha-helix transmembrane protein is the visual pigment of the vertebrate rod photoreceptor cells that mediate dim light vision. In the active binding site of this protein the ligand or chromophore, 11-cis-retinal, is covalently bound via a protonated Schiff base to lysine residue 296. Here we present the complete (1)H and (13)C assignments of the 11-cis-retinylidene chromophore in its ligand-binding site determined with ultra high field magic angle spinning NMR. Native bovine opsin was regenerated with 99% enriched uniformly (13)C-labeled 11-cis-retinal. From the labeled pigment, (13)C carbon chemical shifts could be obtained by using two-dimensional radio frequency-driven dipolar recoupling in a solid-state magic angle spinning homonuclear correlation experiment. The (1)H chemical shifts were assigned by two-dimensional heteronuclear ((1)H-(13)C) dipolar correlation spectroscopy with phase-modulated Lee-Goldburg homonuclear (1)H decoupling applied during the t(1) period. The data indicate nonbonding interactions between the protons of the methyl groups of the retinylidene ionone ring and the protein. These nonbonding interactions are attributed to nearby aromatic acid residues Phe-208, Phe-212, and Trp-265 that are in close contact with, respectively, H-16/H-17 and H-18. Furthermore, binding of the chromophore involves a chiral selection of the ring conformation, resulting in equatorial and axial positions for *CH*(3)-16 and *CH*(3)-17.

L3 ANSWER 15 OF 76 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 2002287645 MEDLINE
DOCUMENT NUMBER: 22022003 PubMed ID: 12024019
TITLE: Leukemia-associated Rho guanine nucleotide exchange factor promotes G alpha q-coupled activation of RhoA.
AUTHOR: Booden Michelle A; Siderovski David P; Der Channing J
CORPORATE SOURCE: Department of Pharmacology, Lineberger Comprehensive Cancer Center, University of North Carolina at Chapel Hill, 27599, USA.. mbooden@med.unc.edu
CONTRACT NUMBER: CA63071 (NCI)
CA92240 (NCI)
GM62338 (NIGMS)
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (2002 Jun) 22 (12) 4053-61.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 20020528
Last Updated on STN: 20020723
Entered Medline: 20020624
AB Leukemia-associated Rho guanine-nucleotide exchange factor *(LARG) belongs* to the subfamily of Dbl homology RhoGEF proteins (including p115 RhoGEF and PDZ-RhoGEF) that possess amino-terminal regulator of G protein signaling (RGS) boxes also found within GTPase-accelerating proteins (GAPs) for heterotrimeric G protein alpha subunits. p115 RhoGEF stimulates the intrinsic GTP hydrolysis activity of G alpha 12/13 subunits and acts as an effector for G13-coupled receptors by linking receptor activation to RhoA activation. The presence of RGS box and Dbl homology domains within LARG suggests this protein may also function as a GAP toward specific G alpha subunits and couple G alpha activation to RhoA-mediating signaling pathways. Unlike the RGS box of p115 RhoGEF, the RGS box of LARG interacts not only with G alpha 12 and G alpha 13 but also with G alpha q. In cellular coimmunoprecipitation studies, the LARG RGS box formed stable complexes with the transition state mimetic forms of G alpha q, G alpha 12, and G alpha 13. Expression of the LARG RGS box diminished the transforming activity of oncogenic ***G*** ***protein*** - ***coupled*** ***receptors*** (***Mas*** , G2A, and m1-muscarinic cholinergic) coupled to G alpha q and G alpha 13. Activated G alpha q, as well as G alpha 12 and G alpha 13, cooperated with LARG and caused synergistic activation of RhoA, suggesting that all three G alpha subunits stimulate LARG-mediated activation of RhoA. Our findings suggest that the RhoA exchange factor LARG, unlike the related p115 RhoGEF and PDZ-RhoGEF proteins, can serve as an effector for Gq-coupled receptors, mediating their functional linkage to RhoA-dependent signaling pathways.

L3 ANSWER 16 OF 76 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 2002072351 MEDLINE
DOCUMENT NUMBER: 21656985 PubMed ID: 11798184
TITLE: Imprinting of the murine ***MAS*** protooncogene is restricted to its antisense RNA.
AUTHOR: Alenina Natalia; Bader Michael; Walther Thomas
CORPORATE SOURCE: Max-Delbruck-Center for Molecular Medicine (MDC), Berlin-Buch, Germany.
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (2002 Jan 25) 290 (3) 1072-8.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200202
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020220
Entered Medline: 20020219
AB The ***Mas*** protooncogene encodes a ***G*** ***protein*** - ***coupled*** ***receptor*** with the common seven transmembrane domains and may be involved in the actions of angiotensins. The gene is *located in close proximity to the paternally imprinted Igf2r gene and its* maternal imprinting has been reported but remained controversial. We used mice carrying a targeted deletion of the ***Mas*** protooncogene on the maternal or paternal chromosome to clarify this issue. In all ***Mas*** -expressing organs of adult mice such as heart, kidney, testis or brain, no ***Mas*** mRNA was missing in heterozygous animals inheriting the deleted allele from the father excluding mono-allelic paternal expression. However, we show exclusive paternal expression of a ***Mas*** antisense RNA, confirming the maternal imprinting of this antisense RNA in all investigated adult tissues and in embryos. Our results strongly suggest that ***Mas*** is not imprinted in mice but that an antisense RNA probably starting in the neighboring Igf2r gene is maternally imprinted in both embryos and adult organs.

L3 ANSWER 17 OF 76 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 2002721178 MEDLINE
DOCUMENT NUMBER: 22371440 PubMed ID: 12482885
TITLE: Activation of a PTX-insensitive G protein is involved in histamine-induced recombinant M-channel modulation.
AUTHOR: Guo Juan; Schofield Geoffery G
CORPORATE SOURCE: Department of Physiology, Tulane University Health Sciences Center, 1430 Tulane Avenue, New Orleans, LA 70112, USA.
SOURCE: JOURNAL OF PHYSIOLOGY, (2002 Dec 15) 545 (Pt 3) 767-81.
Journal code: 0266262. ISSN: 0022-3751.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200307
ENTRY DATE: Entered STN: 20021218
Last Updated on STN: 20030703
Entered Medline: 20030702
AB *The M-type potassium current (I(M))* plays a dominant role in regulating membrane excitability and is modulated by many neurotransmitters. However, except in the case of bradykinin, the signal transduction pathways involved in M-channel modulation have not been fully elucidated. The channels underlying I(M) are produced by the coassembly of KCNQ2 and KCNQ3 channel subunits and can be expressed in heterologous systems where they can be modulated by several neurotransmitter receptors including histamine H(1) receptors. In HEK293T cells, histamine acting via transiently expressed H(1)R produced a strong inhibition of recombinant M-channels but had no overt effects on the voltage dependence or voltage range of I(M) activation. In addition, the modulation of I(M) by histamine was not voltage sensitive, whereas channel gating, particularly deactivation, was accelerated by histamine. Non-hydrolysable guanine nucleotide analogues (GDP-beta-S and GTP-gamma-S) and pertussis toxin (PTX) treatment demonstrated the involvement of a PTX-insensitive G protein in the signal transduction pathway mediating histamine-induced I(M) modulation. Abrogation of the histamine-induced modulation of I(M) by expression of a C-terminal construct of phospholipase C (PLC-beta1-ct), which buffers activated Galpha(q/11) subunits, implicates this G protein alpha subunit in the modulatory pathway. On the other hand, *abrogation of* the histamine-induced modulation of I(M) by expression of two constructs which buffer free betagamma subunits, transducin (Galphat) and a C-terminal construct of a G protein receptor kinase (***MAS*** -GRK2-ct), implicates betagamma dimers in the modulatory pathway. These findings demonstrate that histamine modulates recombinant M-channels in HEK293T cells via a PTX-insensitive G protein, probably Galpha(q/11), in a similar manner to a number of other ***G*** ***protein*** - ***coupled*** ***receptors*** . However, histamine-induced I(M) modulation in HEK293T cells is novel in that betagamma subunits in addition to Galpha(q/11) subunits appear to be involved in the modulation of KCNQ2/3 channel currents.

L3 ANSWER 18 OF 76 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 2002229847 MEDLINE
DOCUMENT NUMBER: 21964182 PubMed ID: 11967280
TITLE: Cell type-specific expression of the ***Mas*** proto-oncogene in testis.
AUTHOR: Alenina Natalia; Baranova Tatjana; Smirnow Eugene; Bader Michael; Lippoldt Andrea; Patkin Eugene; Walther Thomas
CORPORATE SOURCE: Department of Cardiology, University Hospital Benjamin Franklin, Berlin-Buch, Germany.
SOURCE: JOURNAL OF HISTOCHEMISTRY AND CYTOCHEMISTRY, (2002 May) 50 (5) 691-6.
Journal code: 9815334. ISSN: 0022-1554.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 20020423
Last Updated on STN: 20020611
Entered Medline: 20020610
AB The ***Mas*** proto-oncogene encodes a ***G*** - ***protein*** - ***coupled*** ***receptor*** with the common seven transmembrane domains and may be involved in the actions of angiotensins. Because ***Mas*** is highly expressed in testis, we investigated the cell type-specificity and the onset of expression of the gene in this organ. Using an RNase protection assay, it could be shown that neither whole testes nor cultured Sertoli and Leydig cells of 12-day-old mice express ***Mas*** mRNA. ***Mas*** expression is first detected in 18-day-old mice and thereafter increases continuously until 6 months of age. By in situ hybridization, the expression could be localized to Leydig cells and Sertoli cells, the signals being much more pronounced in the former. A weak signal was detected in primary spermatocytes. The strong ontogenetically controlled and cell type-specific expression of this membrane-bound receptor in testis implicates a role for the ***Mas*** proto-oncogene in testis maturation and function.

L3 ANSWER 19 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 2002:982746 SCISEARCH
THE GENUINE ARTICLE: 594LY
TITLE: Evidence that angiotensin-(1-7) is an endogenous ligand

for the ***G*** ***protein*** - ***coupled*** ***receptor*** ***Mas***
AUTHOR: Santos R A (Reprint); Silva A C S E; Maric C; Speth R;
Machado R P; Pinheiro S V; Lopes M T; Mendes E P; Bader M;
Schultheiss H P; Campagnole-Santos M J; Walther T
CORPORATE SOURCE: Univ Fed Minas Gerais, BR-30161 Belo Horizonte, MG,
Brazil; Georgetown Univ, Med Ctr, Washington, DC 20057 USA; Washington State Univ, Pullman, WA 99164 USA; Max
Delbruck Ctr Mol Med, Berlin, Germany; Free Univ Berlin, D-1000 Berlin, Germany
COUNTRY OF AUTHOR: Brazil; USA; Germany
SOURCE: HYPERTENSION, (SEP 2002) Vol. 40, No. 3, pp. 387-387. MA
48.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST,
PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0194-911X.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0

L3 ANSWER 20 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:529637 CAPLUS
DOCUMENT NUMBER: 139:193092
TITLE: Identification of ***G*** ***protein*** - ***coupled*** ***receptor*** genes from the human genome sequence. [Erratum to document cited in CA137:243568]
AUTHOR(S): Takeda, Shigeki; Kadowaki, Shiro; Haga, Tatsuya; Takaesu, Hirotomo; Mitaku, Shigeki
CORPORATE SOURCE: Faculty of Medicine, Department of Neurochemistry,
University of Tokyo, Bunkyo-ku, Tokyo, 113-0033, Japan
SOURCE: FEBS Letters (2002), 523(1-3), 257
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB On page 100, in footnote a of Table 3, the second sentence should read:
"Umami receptor, truncated mGluR4 [21], was categorized in "receptors for
endogenous ligand".". To the list of refs. should be added: [21]
Chardhari, N., Landin, A.M. and Roper, S.D. (2000) Nature Neurosci. 3,
113-119.

L3 ANSWER 21 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 2002:437053 SCISEARCH
THE GENUINE ARTICLE: 551TR
TITLE: Probing the environment of neurotensin whilst bound to the
neurotensin receptor by solid state NMR
AUTHOR: Williamson P T F; Bains S; Chung C; Cooke R; Watts A
(Reprint)
CORPORATE SOURCE: Univ Oxford, Biomembrane Struct Unit, Dept Biochem, S
Parks Rd, Oxford OX1 3QU, England (Reprint); Univ Oxford,
Biomembrane Struct Unit, Dept Biochem, Oxford OX1 3QU, England; ETH Honggerberg, Phys Chem Lab, CH-8093 Zurich,
Switzerland; GlaxoSmithKline Med Res Ctr, Stevenage SG1 2NY, Herts, England
COUNTRY OF AUTHOR: England; Switzerland
SOURCE: FEBS LETTERS, (8 MAY 2002) Vol. 518, No. 1-3, pp. 111-115.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE
AMSTERDAM, NETHERLANDS.
ISSN: 0014-5793.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 27
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB A functionally active analogue of neurotensin, neurotensin(8-13), has
been observed whilst bound to the agonist-binding site of the rat neurotensin receptor by nuclear magnetic resonance (NMR). Through the
application of slow magic angle sample spinning and high-power proton decoupling, sufficient resolution and sensitivity were obtained in the carbon-13 spectrum to allow an assignment of many of the side chain resonances arising from uniformly carbon-13/nitrogen-15-labelled neurotensin(8-13) whilst bound to the neurotensin receptor. Significant perturbations in carbon-13 chemical shift were observed upon the binding
of the neurotensin(8-13) to the receptor. Most importantly significant shifts were observed in both the carboxy terminus and tyrosine side chain
of the neurotensin(8-13), suggesting that these sites are important in the interaction of the neurotensin with the agonist-binding site on the neurotensin receptor. Conversely, no perturbations were observed for the
carbon-13 sites within the guanidinium groups of the arginine side chains,
indicating little interaction with the receptor-binding site, or a shielding of the local environment by the surrounding nitrogen atoms. These NMR observations lend further support to previous structure-activity
studies, site-directed mutagenesis and modelling studies of the agonist-binding site of the neurotensin receptor, from which the same specific residues for which NMR perturbations were observed are important
for neurotensin receptor activation by neurotensin. (C) 2002 Federation of
European Biochemical Societies. Published by Elsevier Science B.V. All rights reserved.

L3 ANSWER 22 OF 76 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:476318 BIOSIS
DOCUMENT NUMBER: PREV200200476318
TITLE: Telemetry unmasks salt resistance in ***Mas*** -deficient mice.
AUTHOR(S): Heringer-Walther, S. (1); Gross, V. (1); Schultheiss, H.-P.
(1); Walther, T. (1)
CORPORATE SOURCE: (1) Department of Cardiology and Pneumology, UKBF, Berlin:
heriwalt@aol.com Germany
SOURCE: Clinical and Experimental Pharmacology and Physiology,
(August, 2002) Vol. 29, No. 8, pp. A99.
http://www.blackwell-science.com/cep. print.
Meeting Info.: 3rd International Amsterdam Mouse Symposium
Amsterdam, Netherlands March 13-15, 2002
ISSN: 0305-1870.
DOCUMENT TYPE: Conference
LANGUAGE: English

L3 ANSWER 23 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:417355 CAPLUS
DOCUMENT NUMBER: 137:243568
TITLE: Identification of ***G*** ***protein*** - ***coupled*** ***receptor*** genes from the human genome sequence
AUTHOR(S): Takeda, Shigeki; Kadowaki, Shiro; Haga, Tatsuya; Takaesu, Hirotomo; Mitaku, Shigeki
CORPORATE SOURCE: Faculty of Medicine, Department of Neurochemistry,
University of Tokyo, Tokyo, 113-0033, Japan
SOURCE: FEBS Letters (2002), 520(1-3), 97-101
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We have identified novel ***G*** ***protein*** - ***coupled***
receptors (GPCRs) with no introns in the coding region from the
human genome sequence: 322 olfactory receptors; 22 taste receptors; 128
registered GPCRs for endogenous ligands; 50 novel GPCR candidates homologous to registered GPCRs for endogenous ligands; and 59 novel GPCR
candidates not homologous to registered GPCRs. The total no. of GPCRs
with and without introns in the human genome was estd. to be approx. 950,
of which 500 are odorant or taste receptors and 450 are receptors for endogenous ligands.
REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 24 OF 76 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:332733 BIOSIS
DOCUMENT NUMBER: PREV200200332733
TITLE: Arachidonic acid release from ***Mas*** -transfected COS
cells can be stimulated by Ang-(1-7), Ang III and Ang IV not involving AT1 and AT2 receptors.
AUTHOR(S): De Buhr, I. (1); Vahl, M. (1); Walther, T. (1)
CORPORATE SOURCE: (1) Department of Cardiology and Pneumology, Free
University of Berlin, Klinikum Benjamin-Franklin, 12000, Berlin Germany
SOURCE: Naunyn-Schmiedeberg's Archives of Pharmacology, (March,
2002) Vol. 365, No. Supplement 1, pp. R69. print.
Meeting Info.: 43rd Spring Meeting of the German Society for Experimental and Clinical Pharmacology and Toxicology Mainz, Germany March 12-14, 2002
ISSN: 0028-1298.
DOCUMENT TYPE: Conference
LANGUAGE: English

L3 ANSWER 25 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 2003:621334 SCISEARCH
THE GENUINE ARTICLE: 613QJ
TITLE: Cardiovascular active angiotensin-(1-7) is an endogenous ligand for the ***G*** ***protein*** - ***coupled*** ***receptor*** ***Mas***
AUTHOR: Walther T (Reprint); Silva A S E; Maric C; Speth R; Machado R P; de Buhr I; Campagnole-Santos M; Lemos V S;
Santos R
CORPORATE SOURCE: Fed Univ, Belo Horizonte, MG, Brazil; Georgetown Univ,
Washington, DC USA; Washington State Univ, Pullman, WA 99164 USA
COUNTRY OF AUTHOR: Brazil; USA
SOURCE: CIRCULATION, (5 NOV 2002) Vol. 106, No. 19, Supp. [S], pp.
49-49. MA 242.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST,
PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0009-7322.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0

L3 ANSWER 26 OF 76 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:332529 BIOSIS
DOCUMENT NUMBER: PREV200200332529
TITLE: Angiotensin-(1-7) is a functional ligand for the ***G*** ***protein*** - ***coupled*** ***receptor*** ***Mas*** .
AUTHOR(S): Walther, T. (1); De Buhr, I. (1); Heringer-Walther, S. (1);
Tschoepe, C. (1); Campagnole-Santos, M.; Schultheiss, H.-P. (1); Santos, R. A. S.
CORPORATE SOURCE: (1) Department of Cardiology and Pneumology, Free
University of Berlin, 12200, Berlin Germany
SOURCE: Naunyn-Schmiedeberg's Archives of Pharmacology, (March,
2002) Vol. 365, No. Supplement 1, pp. R17. print.
Meeting Info.: 43rd Spring Meeting of the German Society for Experimental and Clinical Pharmacology and Toxicology Mainz, Germany March 12-14, 2002
ISSN: 0028-1298.
DOCUMENT TYPE: Conference
LANGUAGE: English

L3 ANSWER 27 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 2002:402468 SCISEARCH
THE GENUINE ARTICLE: 542EQ
TITLE: Angiotensin-(1-7) is a functional ligand for the ***G*** ***protein*** - ***coupled*** ***receptor*** ***Mas***
AUTHOR: Walther T (Reprint); De Buhr I; Heringer-Walther S; Tschope C; Campagnole-Santos M; Schultheiss H P; Santos R
A S
CORPORATE SOURCE: Free Univ Berlin, Dept Cardiol & Pneumol, D-12200 Berlin,
Germany; Univ Fed Minas Gerais, Lab Hypertens, Belo Horizonte, MG, Brazil
COUNTRY OF AUTHOR: Germany; Brazil
SOURCE: NAUNYN-SCHMIEDEBERGS ARCHIVES OF PHARMACOLOGY, (MAR 2002)
Vol. 365, Supp. [1], pp. R17-R17. MA 56.
Publisher: SPRINGER-VERLAG, 175 FIFTH AVE, NEW YORK, NY
10010 USA.
ISSN: 0028-1298.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0

L3 ANSWER 28 OF 76 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:79394 BIOSIS
DOCUMENT NUMBER: PREV200300079394
TITLE: Cardiovascular active angiotensin-(1-7) is an endogenous ligand for the ***G*** ***protcin*** - ***coupled*** ***receptor*** ***Mas*** .
AUTHOR(S): Walther, Thomas (1); Simoes e Silva, Ana; Maric, Christine;
Speth, Robert; Machado, Raquel Pillar; de Buhr, Insa (1); Campagnole-Santos, Maria; Lemos, Virginia Soares; Santos, Robson
CORPORATE SOURCE: (1) Free Univ, Berlin, Germany Germany
SOURCE: Circulation, (November 5 2002) Vol. 106, No. 19 Supplement,
pp. II-49. print.
Meeting Info.: Abstracts from Scientific Sessions Chicago, IL, USA November 17-20, 2002 American Heart Association . ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English

L3 ANSWER 29 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 11
ACCESSION NUMBER: 2002-049265 [06] WPIDS
DOC. NO. CPI: C2002-013825
TITLE: Novel human ***G*** ***protein*** - ***coupled*** ***receptor*** polypeptide that is related to ***MAS*** proto-oncogene receptor subfamily, useful as
model and target for developing human therapeutic agent.
DERWENT CLASS: B04 D16
INVENTOR(S): BEASLEY, E M; CRAVCHIK, A; DI FRANCESCO, V; WEI, M
PATENT ASSIGNEE(S): (PEKE) PE CORP NY; (APPL-N) APPLERA CORP
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001081409	A2	20011101	(200206)*	EN	60

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW
AU 2001053771 A 20011107 (200219)
EP 1280823 A2 20030205 (200310) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
RO SE SI TR
US 2003162946 A1 20030828 (200357)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001081409 A2		WO 2001-US13097	20010424
AU 2001053771 A		AU 2001-53771	20010424
EP 1280823	A2	EP 2001-927304	20010424
		WO 2001-US13097	20010424
US 2003162946 A1	Provisional	US 2000-199149P	20000424
	Cont of	US 2000-633146	20000804
		US 2003-407960	20030409

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001053771 A	Based on	WO 2001081409
EP 1280823 A2	Based on	WO 2001081409

PRIORITY APPLN. INFO: US 2000-633146 20000804; US 2000-199149P 20000424; US 2003-407960 20030409
AN 2002-049265 [06] WPIDS
AB WO 200181409 A UPAB: 20020128
NOVELTY - An isolated human ***G*** ***protein*** - ***coupled*** ***receptor*** (GPCR) polypeptide (I) that is related to ***MAS*** proto-oncogene receptor subfamily, consisting or comprising a fully defined sequence of 289 amino acids (S2) as given in the specification, or its fragment comprising 10 contiguous amino acids, or an amino acid sequence of an allelic variant or ortholog of the amino acid sequence of (S2), is new.
DETAILED DESCRIPTION - An isolated human ***G*** ***protein*** - ***coupled*** ***receptor*** (GPCR) polypeptide (I) consists or comprises of
an amino acid sequence of (S2), an amino acid sequence of an allelic variant or an ortholog of (S2), where the allelic variant or ortholog is encoded by a nucleic acid molecule that hybridizes under stringent conditions to the opposite strand of a nucleic acid molecule having a fully defined sequence of 2099 nucleotides (S1) (transcript) or 5303 nucleotides (S3) (genomic) as given in the specification, a fragment of an amino acid sequence of (S2), comprising 10 contiguous, amino acids.
INDEPENDENT CLAIMS are also included for the following:
(1) an isolated antibody (II) that selectively binds to (I) comprising the amino acid sequence of (S2), its allelic variant or ortholog, or fragment;
(2) an isolated nucleic acid molecule (III) consisting or comprising of a nucleotide sequence that encodes (I) or a nucleotide sequence that is complement of the nucleotide sequence encoding (I);
(3) a gene chip comprising (III) that comprises a nucleotide sequence encoding (I), or its complement;
(4) a transgenic non-human animal comprising (III) that comprises a nucleotide sequence encoding (I), or its complement;
(5) a nucleic acid vector (IV) comprising (III) that comprises a nucleotide sequence encoding (I), or its complement;
(6) a host cell comprising (IV);
(7) preparation of (I);
(8) detecting the presence of (I) in a sample involves contacting the sample with a detection agent that specifically allows detection of the presence of the peptide in the sample and then detecting the presence of the peptide;
(9) detecting the presence of (III) in a sample involves contacting the sample with an oligonucleotide that hybridizes to the nucleic acid molecule under stringent conditions and determining whether an oligonucleotide binds to the nucleic acid molecule in the sample;
(10) a pharmaceutical composition (V) comprising an agent identified that binds to (I) comprising an amino acid sequence of (S2), its allelic variant or ortholog or fragment, and a carrier;
(11) an isolated protease peptide (VI) having an amino acid sequence that shares 70% homology with (S2); and
(12) an isolated nucleic acid molecule (VII) encoding a human protease peptide which shares at least 80% homology with (S1) or (S3).
ACTIVITY - None given.
MECHANISM OF ACTION - Gene therapy; GPCR expression or activity modulator. No supporting data is given.
USE - (I) comprising an amino acid sequence of (S2), its allelic variant or ortholog or fragment, is useful for identifying a modulator of a GPCR polypeptide which involves contacting the peptide with an agent and determining if the agent has modulated the function or activity of the peptide. Preferably, the agent is administered to a host cell comprising an expression vector that expresses the peptide. The method optionally involves contacting a cell expressing the peptide with an agent and determining if the agent has modulated the expression of the peptide. (I) comprising an amino acid sequence of (S2), its allelic variant or ortholog or fragment is also useful for identifying an agent that binds to it which involves contacting the peptide with an agent and assaying the contacted mixture to determine whether a complex is formed with the agent bound to the peptide. (V) is useful for treating a disease or condition mediated by human proteases (all claimed). (I) and (III) can be used as models for the development of human therapeutic targets, aid in the identification of therapeutic proteins and serve as targets for the development of human therapeutic agents. (I) and (III) can be used as a query sequence to perform a search against sequence databases to, for example, identify other family members or related sequences. (I) is used to raise antibodies or to elicit another immune response, as reagent (including the labeled reagent) in assays designed to quantitatively determine levels of the protein (or its binding partner or receptor) in biological fluids, and as markers for tissues in which the corresponding protein is preferentially expressed (either constitutively or at a particular stage of tissue differentiation or development or in a disease state). GPCRs isolated from humans and their human/mammalian orthologs serve as targets for identifying agents for use in mammalian therapeutic applications, e.g., a human drug to modulate the cells or tissues that express the receptor. The receptor polypeptides are useful for biological assays involving any of the known GPCR functions or activities or properties useful for diagnosis and treatment of GPCR-related conditions that are specific for the subfamily of GPCRs. The receptor polypeptides are useful for drug screening assays, to identify compounds that modulate receptor activity of the protein in its natural state, or an altered form that causes a specific disease or pathology associated with the receptor and to screen a compound for the ability to stimulate or inhibit interaction between the receptor protein and a molecule that normally interacts with the receptor protein. (I) is also useful in competition binding assays in methods designed to discover compounds that interact with the receptor. The GPCR proteins are also useful to provide a target for diagnosing a disease or predisposition to a disease medicated by the peptide. They are also useful for pharmacogenomic analysis. (II) can be used to isolate (I) by standard techniques, to facilitate the purification of the natural protein from cells and recombinantly produced protein expressed in host cells, to detect the presence of (I) in cells or tissues to determine the pattern of expression of the protein among various tissues in an organism and over the course of normal development, to detect protein in situ, in vitro, or in a cell lysate or supernatant in order to evaluate the abundance and pattern of expression and to assess abnormal tissue distribution or abnormal expression during development or progression of a biological conditions. (II) can also be used to assess expression in disease states such as in active stages of the disease or in an individual with predisposition toward disease related to the protein's function, particularly in cells and tissues that express the receptor.
Dwg.0/3

L3 ANSWER 30 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 12
ACCESSION NUMBER: 2001-611616 [70] WPIDS
CROSS REFERENCE: 2003-555378 [52]
DOC. NO. CPI: C2001-182803
TITLE: New human ***G*** - ***protein*** ***coupled*** ***receptor*** , useful for identifying specific modulators, potential therapeutic agents, is related to the ***MAS*** proto-oncogene receptor family.
DERWENT CLASS: B04 D16 P14
INVENTOR(S): BEASLEY, E M; CRAVCHIK, A; DI FRANCESCO, V; WANG, A
PATENT ASSIGNEE(S): (PEKE) PE CORP NY
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001072840	A2	20011004	(200170)*	EN	58
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW					
AU 2001052968	A	20011008	(200208)		
EP 1278840	A2	20030129	(200310)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001072840	A2	WO 2001-US9657	20010327
AU 2001052968	A	AU 2001-52968	20010327
EP 1278840	A2	EP 2001-926435	20010327
		WO 2001-US9657	20010327

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001052968 A	Based on	WO 2001072840
EP 1278840 A2	Based on	WO 2001072840

PRIORITY APPLN. INFO: US 2000-629817 20000731; US 2000-192310P 20000327
AN 2001-611616 [70] WPIDS
CR 2003-555378 [52]
AB WO 200172840 A UPAB: 20030813
NOVELTY - Isolated peptide (I), comprising, a 312 residue amino acid sequence (S2), fully defined in the specification, an allelic variant or ortholog of (S2) encoded by a nucleic acid that hybridizes under stringent conditions to the complementary strand of a 939 (cDNA) (S1) or 1775 (genomic) (S3) base pair sequence, fully defined in the specification, or at least 10 contiguous residues of (S2), is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) isolated antibodies (Ab) that bind selectively to (I);
(2) isolated nucleic acid (II) that consists of, or comprises, a sequence that encodes (I), or is the complement of it;
(3) a gene chip containing (II);
(4) transgenic non-human animal containing (II);
(5) vector containing (II);
(6) host cell containing the vector of (5);
(7) recombinant production of (I) by expressing (II) in a host cell;
(8) detecting (I) in a sample by reaction with a specific detection agent;
(9) detecting (II) in a sample by reaction with an oligonucleotide that hybridizes to it under stringent conditions;
(10) identifying an agent (A) that modulates, or binds to, (I);
(11) pharmaceutical composition containing (A) and a carrier;
(12) identifying agents that modulate expression of (I);
(13) isolated human protease at least 70 % homologous with (S2); and
(14) isolated nucleic acid encoding a human protease and at least 80% homologous with (S1) or (S3).
ACTIVITY - None given.
No biological data is given.
MECHANISM OF ACTION - Antagonism or agonism of a ***G*** - ***protein*** ***coupled*** ***receptor*** , related to the ***MAS*** proto-oncogene receptor subfamily.
USE - (I) is useful as target for identifying specific modulators and binding agents, potentially useful as human therapeutic agents, especially for control of diseases in which (I) is implicated, and for production of specific antibodies (Ab), or to elicit other immune responses. (I) can also be used as reagents for determination of the level of (I) or its binding partners, as tissue marker, as therapeutics and for pharmacogenomic studies. Ab are useful for isolation and purification o (I), and for determination of (I), in diagnosis and monitoring. Ab is used in pharmacogenomic analysis, for tissue typing and as therapeutic modulators of (I). Nucleic acid (II) that encodes (I) is useful for recombinant expression of (I), and as source of primers and probes (for diagnosis) and of antisense sequences and ribozymes (for therapy). They can be used identifying modulators of its expression, monitoring gene expression during therapy, identifying mutations in the (I)-encoding gene, construction of gene therapy vectors, and preparing transgenic animals, used to study function of (I) and to identify modulators.
Dwg.0/3

L3 ANSWER 31 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 13
ACCESSION NUMBER: 2002-055135 [07] WPIDS
DOC. NO. CPI: C2002-015674
TITLE: New human ***G*** - ***protein*** ***coupled*** ***receptor*** , useful for identifying specific modulators, potential anticancer agents, is related to the ***MAS*** proto-oncogene receptor family.
DERWENT CLASS: B04 D16
INVENTOR(S): BEASLEY, E M; CRAVCHIK, A; DI FRANCESCO, V; WEI, M
PATENT ASSIGNEE(S): (PEKE) PE CORP NY
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001072838	A2	20011004	(200207)*	EN	62
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW					
AU 2001047766	A	20011008	(200208)		
EP 1278839	A2	20030129	(200310)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
JP 2003528634	W	20030930	(200365)		89

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

WO 2001072838 A2 WO 2001-US9523 20010327
AU 2001047766 A AU 2001-47766 20010327
EP 1278839 A2 EP 2001-920742 20010327
WO 2001-US9523 20010327
JP 2003528634 W JP 2001-571769 20010327
WO 2001-US9523 20010327

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001047766 A	Based on	WO 2001072838
EP 1278839	A2 Based on	WO 2001072838
JP 2003528634 W	Based on	WO 2001072838

PRIORITY APPLN. INFO: US 2000-635593 20000809; US 2000-192853P 20000329
AN 2002-055135 [07] WPIDS
AB WO 200172838 A UPAB: 20020130
NOVELTY - Isolated peptide (I) comprising, a 330 residue amino acid sequence (S2), fully defined in the specification, an allelic variant or ortholog of (S2) encoded by a nucleic acid that hybridizes under stringent conditions to the complementary strand of a 993 (cDNA) (S1) or 11046 (genomic) (S3), base pair sequence, fully defined in the specification, or at least 10 contiguous residues of (S2), is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) isolated antibodies (Ab) that bind selectively to (I);
(2) isolated nucleic acid (II) that consists of, or comprises, a sequence that encodes (I) or it's complement;
(3) a gene chip containing (II);
(4) transgenic non-human animal containing (II);
(5) vector containing (II);
(6) host cell containing the vector of (4);
(7) recombinant production of (I) by expressing (II) in a host cell;
(8) detecting (I) in a sample by reaction with a specific detection agent;
(9) detecting (II) in a sample by reaction with an oligonucleotide that hybridizes to it under stringent conditions;
(10) identifying an agent (A) that modulates, or binds to, (I);
(11) pharmaceutical composition containing (A) and a carrier;
(12) identifying agents that modulate expression of (I);
(13) isolated human protease at least 70 % homologous with (S2); and
(14) isolated nucleic acid encoding a human protease and at least 80 % homologous with (S1) or (S3).
ACTIVITY - Cytostatic.
No biological data is given.
MECHANISM OF ACTION - Antagonism or agonism of a ***G*** - ***protein*** ***coupled*** ***receptor*** , related to the ***MAS*** proto-oncogene receptor family.
USE - (I) is useful as target for identifying specific modulators and binding agents, potentially useful as human therapeutic agents, especially for control of tumors that express (I), and for production of specific antibodies (Ab), or to elicit other immune responses. (I) is used as reagents for determination of the level of (I) or its binding partners, as tissue marker, as therapeutics, and for pharmacogenomic studies. Ab are useful for isolation and purification of (I), and for determination of (I), in diagnosis and monitoring. AB are also used in pharmacogenomic analysis, for tissue typing, and as therapeutic modulators of (I). Nucleic acid (II) that encodes (I) is useful for recombinant expression of (I), and as source of primers and probes (for diagnosis) and of antisense sequences and ribozymes (for therapy). They can be used for identifying modulators of its expression, monitoring gene expression during therapy, identifying mutations in the (I)-encoding gene, construction of gene therapy vectors, and preparing transgenic animals, used to study function of (I) and to identify modulators.
Dwg.0/3

L3 ANSWER 32 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 14
ACCESSION NUMBER: 2001-639125 [73] WPIDS
DOC. NO. CPI: C2001-189063
TITLE: Polynucleotide encoding ***mas*** oncogene-related ***G*** - ***protein*** ***coupled*** ***receptor*** for treating neurological disorders, cardiovascular disorders, Parkinson's disease, cancer, anorexia, bulimia and asthma.
DERWENT CLASS: B04 D16
INVENTOR(S): RAMAKRISHNAN, S
PATENT ASSIGNEE(S): (FARB) BAYER AG; (RAMA-I) RAMAKRISHNAN S
COUNTRY COUNT: 96
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001070971	A2	20010927	(200173)*	EN	75

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ
LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD
SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001054732 A 20011003 (200210)
EP 1268795 A2 20030102 (200310) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
RO SE SI TR
US 2003049787 A1 20030313 (200321)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001070971	A2	WO 2001-EP3340	20010323
AU 2001054732	A	AU 2001-54732	20010323
EP 1268795	A2	EP 2001-927791	20010323
		WO 2001-EP3340	20010323
US 2003049787	A1	WO 2001-EP3340	20010323
		US 2002-239028	20020918

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001054732	A Based on	WO 2001070971
EP 1268795	A2 Based on	WO 2001070971

PRIORITY APPLN. INFO: US 2000-191719P 20000324
AN 2001-639125 [73] WPIDS
AB WO 200170971 A UPAB: 20011211
NOVELTY - An isolated polynucleotide (I) encoding a ***mas*** oncogene-related ***G*** - ***protein*** ***coupled*** ***receptor*** polypeptide (II) comprising a sequence having at least 50% identity to a sequence (S1) comprising 167 amino acids fully defined in the specification or S1, and comprising a sequence (S2) of 503 nucleotides fully defined in the specification, is new.
DETAILED DESCRIPTION - An isolated polynucleotide (I) encoding a ***mas*** oncogene-related ***G*** - ***protein*** ***coupled*** ***receptor*** polypeptide (II) comprising a sequence having at least 50% identity to a sequence (S1) comprising 167 amino acids fully defined in the specification or S1, and comprising a sequence (S2) of 503 nucleotides fully defined in the specification, is new. (I) comprises a sequence encoding (II), a sequence comprising S2, a sequence which hybridizes under stringent conditions to the above said sequences, a sequence which deviated from the above said sequences due to the degeneration of genetic code, or a fragment, derivative or allelic variant of the above said polynucleotide sequences.
INDEPENDENT CLAIMS are also included for the following:
(1) an expression vector (III) containing (I);
(2) a host cell (IV) containing (III);
(3) a substantially purified ***mas*** oncogene-related ***G*** - ***protein*** ***coupled*** ***receptor*** polypeptide (II) encoded by (I);
(4) producing (II);
(5) detecting (I) or (II), by contacting a biological sample with a reagent which specifically interacts with (I) or (II);
(6) a diagnostic kit for conducting the above said method;
(7) reducing the activity of (II), by contacting a cell with a reagent which specifically binds to (I) or (II), such that the activity of (II) is reduced;
(8) a reagent (R) that modulates the activity of (I) or (II), identified by the above said method; and
(9) a pharmaceutical composition (PC) comprising (III) or (R).
ACTIVITY - Antiparkinsonian; antibacterial; fungicide; protozoacide; virucide; analgesic; cytostatic; antiasthmatic; cardiant; hypotensive; osteopathic; antianginal; antiulcer; antiallergic; neuroprotectant; anti-HIV; tranquilizer; neuroleptic; antimanic; antidepressant; nootropic; anticonvulsant.
MECHANISM OF ACTION - Regulates ***mas*** oncogene-related-GPCR; antisense gene therapy.
Antisense ***mas*** oncogene-related-GPCR oligonucleotides comprising at least 11 contiguous nucleotides of (I) was administered to a patient with breast tumor. The size of the patient's breast tumor was found to be decreased.
USE - (I) Is useful for detecting a polynucleotide encoding a ***mas*** oncogene-related ***G*** - ***protein*** ***coupled*** ***receptor*** (GPCR) polypeptide in a biological sample, by hybridizing (I) to a nucleic acid material of a biological sample, to form a hybridization complex, and detecting the hybridization complex. Preferably, the nucleic acid material of the biological sample is amplified before hybridization. (II) is useful for screening agents which decrease the activity of ***mas*** oncogene-related GPCR, by contacting a test compound with any ***mas*** oncogene-related GPCR polypeptide encoded by (I), and detecting the binding of test compound with ***mas*** oncogene-related GPCR polypeptide, where a test compound which binds to the polypeptide is identified as a potential therapeutic agent for decreasing the activity of ***mas*** oncogene-related GPCR.
(II) is useful for screening agents which regulate the activity of ***mas*** oncogene-related GPCR, by contacting a test compound with a ***mas*** oncogene-related GPCR polypeptide encoded by (I), and detecting ***mas*** oncogene-related GPCR activity of the polypeptide, where a test compound that increases the ***mas*** oncogene-related GPCR activity is identified as a potential therapeutic agent for increasing the activity of the polypeptide, and where a test compound that decreases activity of the polypeptide is identified as a potential therapeutic agent for decreasing the activity of the polypeptide. (I) is useful for screening agents which decrease the activity of ***mas*** oncogene-related GPCR, by contacting a test compound with (I), and detecting binding of the test compound to the polynucleotide, where a test compound which binds to the polynucleotide is identified as a potential therapeutic agent for decreasing the activity of ***mas*** oncogene-related GPCR.
PC is useful for modulating the activity of ***mas*** oncogene-related GPCR in a disease, e.g. bacterial, fungal, protozoan and viral infection, pain, cancer, anorexia, bulimia, asthma, Parkinson's disease, acute heart failure, hypertension, hypotension, urinary retention, osteoporosis, angina pectoris, myocardial infarction, ulcer, allergy, multiple sclerosis, benign prostatic hypertrophy, psychotic and neurological disorders, mental retardation, dyskinesia, neoplasia, cardiovascular disorder and seizure disorder (claimed), and also HIV infections, anxiety, schizophrenia, manic depression, delirium, dementia Huntington's disease and Tourette's syndrome.
(II) is useful as a bait protein in a two-hybrid assay or a three-hybrid assay, to identify other proteins which bind to or interact with ***mas*** oncogene-related GPCR polypeptide and modulate its activity. (II) is useful for raising antibodies which can block the receptor and prevent the ligand binding.
Dwg.0/0

L3 ANSWER 33 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 15
ACCESSION NUMBER: 2001-602745 [68] WPIDS
DOC. NO. NON-CPI: N2001-449756
DOC. NO. CPI: C2001-178578
TITLE: Novel ***mas*** receptor-analogous proteins, comprising ***G*** ***protein*** - ***coupled*** ***receptor*** protein originating in mouse heart and whole rat brain, for use in diagnosis and developing drugs for treatment of e.g. hypertension and cancer.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): FUKUSUMI, S; HINUMA, S
PATENT ASSIGNEE(S): (TAKE) TAKEDA CHEM IND LTD; (FUKU-I) FUKUSUMI S; (HINU-I) HINUMA S
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001068847	A1	20010920	(200168)*	JA	116

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001041153 A 20010924 (200208)
JP 2002238584 A 20020827 (200271) 43
US 2003082648 A1 20030501 (200331)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001068847	A1	WO 2001-JP2053	20010315
AU 2001041153	A	AU 2001-41153	20010315
JP 2002238584	A	JP 2001-74256	20010315
US 2003082648	A1	WO 2001-JP2053	20010315
		US 2002-221841	20020912

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001041153	A Based on	WO 2001068847

PRIORITY APPLN. INFO: JP 2000-381698 20001211; JP 2000-81835 20000317
AN 2001-602745 [68] WPIDS
AB WO 200168847 A UPAB: 20011121
NOVELTY - A protein which has an amino acid sequence identical or substantially similar to (I), or its salt, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:
(1) a partial peptide of the protein, or its amide, ester or their salt;
(2) a polynucleotide containing a polynucleotide encoding the protein or its partial peptide;
(3) a recombinant vector containing the polynucleotide;
(4) a transformant which is transformed with the recombinant vector;
(5) a process for producing the protein, its partial peptide or its amide, ester, or their salt which is by culturing the transformant to produce the protein or its partial peptide;
(6) an antibody that is for the protein, its partial peptide or its amide, ester, or their salt;
(7) diagnostics containing the antibody;
(8) ligands against the protein, its partial peptide or its amide, ester, or their salt which are obtained by using them;
(9) drugs containing the ligands;
(10) a method for determining ligands against the protein, its partial peptide or its amide, ester, or their salt which is by using them;

(11) screening compounds or their salts that can alter the binding activity between the ligand and the protein, its partial peptide or its amide, ester, or their salt by using them;
(12) a kit for screening compounds or their salts that can alter the binding activity between the ligand and the protein, its partial peptide or its amide, ester, or their salt containing them;
(13) compounds or their salts thus screened;
(14) drugs containing these compounds or their salts;
(15) a polynucleotide hybridizable with the polynucleotide under stringent conditions;
(16) a polynucleotide containing a base sequence complementary to the
polynucleotide or a part of it;
(17) quantitating mRNA of the protein which is by using the protein-encoded polynucleotide or a part of it;
(18) quantitating the protein, its partial peptide, its amide, ester or their salt which is by using the antibody;
(19) diagnosing diseases related to function of the protein by using any of the above quantification methods;
(20) screening compounds or their salts that can alter expression dose of the protein which is by using any of the above quantification methods; and
(21) screening compounds or their salts that can alter the amount of protein on cell membrane by using any of the above quantification methods.
ACTIVITY - Hypotensive; immunosuppressive; cytostatic.
MECHANISM OF ACTION - Signal transducer.
USE - The proteins and encoded DNAs are useful in diagnosis and developing drugs for treatment of e.g. hypertension, autoimmune diseases
and cancer, including by gene therapy.
Dwg.0/4

L3 ANSWER 34 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2001-611617 [70] WPIDS
DOC. NO. CPI: C2001-182804
TITLE: New human ***G*** - ***protein*** ***coupled***
receptor , useful for identifying specific modulators, potential therapeutic agents, is related to the aminergic receptor family.
DERWENT CLASS: B04 D16
INVENTOR(S): BEASLEY, E M; CRAVCHIK, A; DI FRANCESCO, V; KODIRA, C
PATENT ASSIGNEE(S): (PEKE) PE CORP NY
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001072841	A2	20011004	(200170)*	EN	60

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW
AU 2001051000 A 20011008 (200208)
EP 1278775 A2 20030129 (200310) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
RO SE SI TR
JP 2003528636 W 20030930 (200365) 87

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001072841	A2	WO 2001-US9660	20010327
AU 2001051000	A	AU 2001-51000	20010327
EP 1278775	A2	EP 2001-924338	20010327
		WO 2001-US9660	20010327
JP 2003528636	W	JP 2001-571772	20010327
		WO 2001-US9660	20010327

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001051000	A Based on	WO 2001072841
EP 1278775	A2 Based on	WO 2001072841
JP 2003528636	W Based on	WO 2001072841

PRIORITY APPLN. INFO: US 2000-633145 20000804; US 2000-192311P 20000327
AN 2001-611617 [70] WPIDS
AB WO 200172841 A UPAB: 20011129
NOVELTY - Isolated peptide (I), comprising a 338 residue amino acid sequence (S2), fully defined in the specification, an allelic variant or ortholog of (S2) encoded by a nucleic acid that hybridizes under stringent
conditions to the complementary strand a 1017 (cDNA) or 3501 base pair
sequence (S1), fully defined in the specification (genomic), or a fragment
of (S2) of at least 10 contiguous residues, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the
following:
(1) isolated antibodies (Ab) that bind selectively to (I);
(2) isolated nucleic acid (II) that consists of, or comprises, a sequence that encodes (I) or it's complement;
(3) a gene chip containing (II);
(4) transgenic non-human animal containing (II);
(5) vector containing (II);
(6) host cell containing the vector of (5);
(7) recombinant production of (I) by expressing (II) in a host cell;
(8) detecting (I) in a sample by reaction with a specific detection agent;
(9) detecting (II) in a sample by reaction with an oligonucleotide that hybridizes to it under stringent conditions;
(10) identifying an agent (A) that modulates, or binds to, (I);
(11) pharmaceutical composition containing (A) and a carrier;
(12) identifying agents that modulate expression of (I);
(13) isolated human protease at least 70 % homologous with (S2); and
(14) isolated nucleic acid encoding a human protease and at least 80 % homologous with (S1).
ACTIVITY - None given.
MECHANISM OF ACTION - Antagonism or agonism of a ***G*** - ***protein*** ***coupled*** ***receptor*** , related to the aminergic receptor subfamily.
No biological data is given.
USE - (I) is useful as target for identifying specific modulators and binding agents, potentially useful as human therapeutic agents, especially
for control of diseases in which (I) is implicated. (I) is also useful for production of specific antibodies (Ab), or to elicit other immune responses, and as reagents for (diagnostic) determination of the level of (I) or its binding partners. (I) can be used as tissue marker, as therapeutics, and for pharmacogenomic studies. Ab are useful for isolation
and purification of (I), and for determination of (I), in diagnosis and monitoring. AB can be used in pharmacogenomic analysis, for tissue typing,
and as therapeutic modulators of (I). Nucleic acid (II) that encodes (I) is useful for recombinant expression of (I), and as source of primers and probes (for diagnosis) and of antisense sequences and ribozymes (for therapy). It can also be used for identifying modulators of its expression, monitoring gene expression during therapy, identifying mutations in the (I)-encoding gene, construction of gene therapy vectors,
and preparing transgenic animals, used to study function of (I) and to identify modulators.
Dwg.0/3

L3 ANSWER 35 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2001-611615 [70] WPIDS
DOC. NO. NON-CPI: N2001-456522
DOC. NO. CPI: C2001-182802
TITLE: New human ***G*** - ***protein*** ***coupled***
receptor , useful for identifying specific modulators, potential therapeutic agents, is related to the neurotransmitter receptor family.
DERWENT CLASS: B04 D16 P14 S03
INVENTOR(S): BEASLEY, E M; CRAVCHIK, A; DI FRANCESCO, V; WANG, A
PATENT ASSIGNEE(S): (PEKE) PE CORP NY; (APPL-N) APPLERA CORP
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001072839	A2	20011004	(200170)*	EN	63

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW
AU 2001047767 A 20011008 (200208)
EP 1278774 A2 20030129 (200310) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
RO SE SI TR
US 2003113789 A1 20030619 (200341)
JP 2003528635 W 20030930 (200365) 91

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001072839	A2	WO 2001-US9524	20010327
AU 2001047767	A	AU 2001-47767	20010327
EP 1278774	A2	EP 2001-920743	20010327
		WO 2001-US9524	20010327
US 2003113789	A1 Provisional	US 2000-192326P	20000327
	Cont of	US 2000-637603	20000815
		US 2002-330220	20021230
JP 2003528635	W	JP 2001-571770	20010327
		WO 2001-US9524	20010327

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001047767	A Based on	WO 2001072839
EP 1278774	A2 Based on	WO 2001072839
JP 2003528635	W Based on	WO 2001072839

PRIORITY APPLN. INFO: US 2000-637603 20000815; US 2000-192326P 20000327; US 2002-330220 20021230
AN 2001-611615 [70] WPIDS
AB WO 200172839 A UPAB: 20030919
NOVELTY - Isolated peptide (I), comprising a 342 residue amino acid sequence (S2), fully defined in the specification, an allelic variant or ortholog of (S2) encoded by a nucleic acid that hybridizes under stringent
conditions to the complementary strand of a 1029 (cDNA) (S1) or 6884 (genomic) (S3) base pair sequence, fully defined in the specification, or at least 10 contiguous residues of (S2), is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the
following:
(1) isolated antibodies (Ab) that bind selectively to (I);
(2) isolated nucleic acid (II) that consists of, or comprises, a sequence that encodes (I), or it's complement;
(3) a gene chip containing (II);
(4) transgenic non-human animal containing (II);
(5) vector containing (II);
(6) host cell containing the vector of (5);
(7) recombinant production of (I) by expressing (II) in a host cell;
(8) detecting (I) in a sample by reaction with a specific detection agent;
(9) detecting (II) in a sample by reaction with an oligonucleotide that hybridizes to it under stringent conditions;
(10) identifying an agent (A) that modulates, or binds to, (I);
(11) pharmaceutical composition containing (A) and a carrier;
(12) identifying agents that modulate expression of (I);
(13) isolated human protease at least 70 % homologous with (S2); and
(14) isolated nucleic acid encoding a human protease and at least 80 % homologous with (S1) or (S3).
ACTIVITY - None given.
No biological data is given.
MECHANISM OF ACTION - Antagonism or agonism of a ***G*** - ***protein*** ***coupled*** ***receptor*** , related to the neurotransmitter receptor subfamily.
USE - (I) is useful as target for identifying specific modulators and binding agents, potentially useful as human therapeutic agents, especially
for control of diseases in which (I) is implicated, and for production of specific antibodies (Ab), or to elicit other immune responses. (I) ,may be
used as reagents for determination of the level of (I) or its binding partners, as tissue marker, as therapeutics and for pharmacogenomic studies. Ab are useful for isolation and purification of (I), and for determination of (I), in diagnosis and monitoring. Ab can be used in pharmacogenomic analysis, for tissue typing, and as therapeutic modulators
of (I). Nucleic acid (II) that encodes (I) is useful for recombinant expression of (I), and as source of primers and probes (for diagnosis) and
of antisense sequences and ribozymes (for therapy). They can be used for
identifying modulators of its expression, monitoring gene expression during therapy, identifying mutations in the (I)-encoding gene, construction of gene therapy vectors and preparing transgenic animals, used to study function of (I) and to identify modulators.
Dwg.0/3

L3 ANSWER 36 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:507951 CAPLUS
DOCUMENT NUMBER: 135:87148
TITLE: Metal ion binding site-based method of identifying ligands of biological target molecules for drug discovery
INVENTOR(S): Elling, Christian E.; Gerlach, Lars Ole; Holst Lange, Birgitte; Pedersen, Jan Torleif; Schwartz, Thue W.
PATENT ASSIGNEE(S): 7TM Pharma, Den.
SOURCE: PCT Int. Appl., 114 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001050127	A2	20010712	WO 2000-EP13389	20001229
WO 2001050127	A3	20020131		
WO 2001050127	C2	20020912		

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH,
CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI,
GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR,
KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ,
NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR,
TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD,
RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
US 2002061599 A1 20020523 US 2000-752102 20001229
EP 1242824 A2 20020925 EP 2000-993741 20001229
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
WO 2002054077 A2 20020711 WO 2001-DK867 20011221
W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY,

BZ, CA, CH,
CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES,
FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG,
KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW,
MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK,
SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM,
AZ, BY, KG, KZ
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: DK 1999-1879 A 19991230
DK 1999-1880 A 19991230
US 2000-175401P P 20000111
US 2000-175994P P 20000111
DK 2000-705 A 20000428
US 2000-202990P P 20000509
WO 2000-EP13389 W 20001229
DK 2001-536 A 20010330
US 2001-280237P P 20010330
OTHER SOURCE(S): MARPAT 135:87148
AB The invention provides a mol. approach for rapidly and selectively identifying small org. mol. ligands, i.e. compds., that are capable of interacting with and binding to specific sites on biol. target mols. The methods of the invention are applicable to any biol. target mol. that has or can be manipulated to have a metal-ion binding site. Biol. target mols. are e.g. proteins, polypeptides, oligopeptides, nucleic acids, carbohydrates, nucleoproteins, glycoproteins, glycolipids, lipoproteins and derivs. thereof. More specifically, the biol. target mols. include membrane receptors, signal transduction proteins, scaffolding proteins, nuclear receptors, steroid receptors, intracellular receptors, transcription factors, enzymes, allosteric enzyme regulatory proteins, growth factors, hormones, neuropeptides and Igs. A very interesting group of biol. target mols. are membrane proteins such as, e.g., transmembrane protein (e.g. 7 TMs). The methods described herein make it possible to construct and screen libraries of compds. specifically directed against predetd. epitopes on the biol. target mols. The compds. are initially constructed to be bifunctional, i.e. having both a metal-ion binding moiety, which conveys them with the ability to bind to either a natural or an artificially constructed metal-ion binding site as well as a variable moiety, which is varied chem. to probe for interactions with specific parts of the biol. target mol. located spatially adjacent to the metal-ion binding site. Compds. may subsequently be further modified to bind to the unmodified biol. target mol. without help of the bridging metal-ion. The methods according to the invention may be performed easily and quickly and lead to unambiguous results. The compds. identified by the methods may themselves be employed for various applications or may be further derivatized or modified to provide novel compds. The methodol. of the invention is useful in drug discovery.

L3 ANSWER 37 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:507755 CAPLUS
DOCUMENT NUMBER: 135:103476
TITLE: cDNA and protein sequence of mouse ***G*** ***protein*** - ***coupled*** ***receptor*** , ***mas*** and their uses in drug screening, diagnosis and therapeutics
INVENTOR(S): Lane, Pamela; Tsui, Ping; Elshourbagy, Nabil
PATENT ASSIGNEE(S): SmithKline Beecham Corporation, USA; SmithKline Beecham PLC
SOURCE: PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001049744	A1	20010712	WO 2001-US54	20010103

W: GB, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR
PRIORITY APPLN. INFO.: US 2000-174499P P 20000104
US 2000-742508 A 20001221
AB This invention provides cDNA and protein sequence of mouse ***G*** ***protein*** - ***coupled*** ***receptor*** , ***mas*** . Also disclosed are methods for screening for compds. that either agonize or antagonize Mus musculus ***mas*** . Such compds. are expected to be useful in treatment of human diseases assocd. with ***G*** ***protein*** - ***coupled*** ***receptor*** disorder. These diseases include, but are not limited to infection with bacteria, fungi, protozoa and viruses including HIV-1 and HIV-2; pain; cancer; diabetes; obesity; anorexia; bulimia; asthma; Parkinson's disease; acute heart failure; hypotension; hypertension; urinary retention; osteoporosis; angina pectoris; myocardial infarction; stroke; ulcers; allergy; benign prostatic hypertrophy; migraine; vomiting; psychotic and neurol. disorders including anxiety, schizophrenia, manic depression, depression, delirium, dementia, and severe mental retardation; and dyskinesias including Huntington's disease or Tourette's syndrome.
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 38 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:380642 CAPLUS
DOCUMENT NUMBER: 135:1263
TITLE: Protein and cDNA sequences of mouse ***G*** ***protein*** - ***coupled*** ***receptor*** AXOR45, and uses thereof in therapy, diagnosis, and drug screening
INVENTOR(S): Trinh, Han Ngoc; Gattu, Mahanandeeshwar
PATENT ASSIGNEE(S): Smithkline Beecham Corporation, USA; Smithkline Beecham Plc
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001036480	A1	20010525	WO 2000-US31835	20001120

W: JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR
PRIORITY APPLN. INFO.: US 1999-166259P P 19991118
US 2000-713981 A 20001116
AB This invention provides protein and cDNA sequences for a newly identified mouse protein, designated AXOR45, which is believed to be a ***G*** ***protein*** ***coupled*** ***receptor*** since it shows homol. with human ***MAS*** proto-oncogene. In one embodiment, the invention relates to drug screening assays of using AXOR45 protein in identifying compds. that may be agonists or antagonists that are potentially useful in therapy. Also disclosed are methods for utilizing AXOR45 polypeptides and polynucleotides in the diagnosis and treatment of diseases assocd. with inappropriate AXOR45 activity or levels.

L3 ANSWER 39 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:168032 CAPLUS
DOCUMENT NUMBER: 134:218017
TITLE: Protein and cDNA sequences of mouse ***G*** ***protein*** - ***coupled*** ***receptor*** CanoMan, and uses thereof
INVENTOR(S): Tsui, Ping; Vawter, Lisa
PATENT ASSIGNEE(S): Smithkline Beecham Corporation, USA; Smithkline Beecham P.L.C.
SOURCE: PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001016177	A1	20010308	WO 2000-US23441	20000825
WO 2001016177	C2	20020906		

W: JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1999-151038P P 19990827
US 2000-644261 A 20000823
AB This invention provides protein and cDNA sequences for a newly identified mouse protein, designated CanoMan, which is believed to be a ***G*** ***protein*** ***coupled*** ***receptor*** since it shows homol. with human ***mas*** oncogene. In one embodiment, the invention relates to drug screening assays of using CanoMan protein in identifying compds. that may be agonists or antagonists that are potentially useful in therapy. Also disclosed are methods for utilizing CanoMan polypeptides and polynucleotides in the diagnosis of diseases assocd. with inappropriate CanoMan activity or levels.
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 40 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:168016 CAPLUS
DOCUMENT NUMBER: 134:218014
TITLE: Protein and cDNA sequences of human ***G*** ***protein*** - ***coupled*** ***receptor*** TheAnt, and uses thereof
INVENTOR(S): Tsui, Ping; Vawter, Lisa
PATENT ASSIGNEE(S): Smithkline Beecham Corporation, USA
SOURCE: PCT Int. Appl., 38 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 11
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001016159	A1	20010308	WO 2000-US23475	20000825

W: JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
US 2001025099 A1 20010927 US 2001-826508 20010405
PRIORITY APPLN. INFO.: US 1999-384610 A 19990827
US 1998-75307P P 19980220
US 1998-75464 A1 19980508
US 1998-75468 A1 19980508
US 1998-144779 A1 19980901
US 1998-188837 A1 19981109
US 1998-193212 A1 19981117
US 1999-253216 A1 19990219
US 1999-260360 A1 19990301
US 1999-274080 A1 19990322
US 1999-287034 A1 19990406
US 1999-328603 A1 19990609
US 1999-337105 B1 19990621
US 1999-363203 A1 19990729
US 1999-425406 A1 19991022
AB This invention provides protein and cDNA sequences for a newly identified human protein, designated TheAnt, which is believed to be a ***G*** ***protein*** ***coupled*** ***receptor*** since it shows homol. with human ***mas*** oncogene. In one embodiment, the inventior relates to drug screening assays of using TheAnt protein in identifying compds. that may be agonists or antagonists that are potentially useful in therapy. Also disclosed are methods for utilizing TheAnt polypeptides and polynucleotides in the diagnosis of diseases assocd. with inappropriate TheAnt activity or levels.
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 41 OF 76 MEDLINE on STN DUPLICATE 16
ACCESSION NUMBER: 2001165709 MEDLINE
DOCUMENT NUMBER: 21159129 PubMed ID: 11258947
TITLE: Ultra-high-field ***MAS*** NMR assay of a multispin labeled ligand bound to its G-protein receptor target in the natural membrane environment: electronic structure of the retinylidene chromophore in rhodopsin.
AUTHOR: Verhoeven M A; Creemers A F; Bovee-Geurts P H; De Grip W J; Lugtenburg J; de Groot H J
CORPORATE SOURCE: Leiden Institute of Chemistry, Gorlaeus Laboratories, Leiden University, P.O. Box 9502, NL-2300 RA Leiden, The Netherlands.
SOURCE: BIOCHEMISTRY, (2001 Mar 20) 40 (11) 3282-8.
Journal code: 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010611
Entered Medline: 20010607
AB 11-Z-[8,9,10,11,12,13,14,15,19,20-(13)C10]Retinal prepared by total synthesis is reconstituted with opsin to form rhodopsin in the natural lipid membrane environment. The 13C shifts are assigned with magic angle spinning NMR dipolar correlation spectroscopy in a single experiment and compared with data of singly labeled retinylidene ligands in detergent-solubilized rhodopsin. The use of multispin labeling in combination with 2-D correlation spectroscopy improves the relative accuracy of the shift measurements. We have used the chemical shift data to analyze the electronic structure of the retinylidene ligand at three levels of understanding: (i) by specifying interactions between the 13C-labeled ligand and the ***G*** - ***protein*** - ***coupled*** ***receptor*** target, (ii) by making a charge assessment of the protonation of the Schiff base in rhodopsin, and (iii) by evaluating the total charge on the carbons of the retinylidene chromophore. In this way it is shown that a conjugation defect is the predominant ground-state property governing the molecular electronics of the retinylidene chromophore in rhodopsin. The cumulative chemical shifts at the odd-numbered carbons (Delta(sigma)odd) of 11-Z-protonated Schiff base models relative to the unprotonated Schiff base can be used to measure the extent of delocalization of positive charge into the polyene. For a series of 11-Z-protonated Schiff base models and rhodopsin, Delta(sigma)odd appears to correlate linearly with the frequency of maximum visible absorption. Since rhodopsin has the largest value of Delta(sigma)odd, the data contribute to existing and converging spectroscopic evidence for a complex counterion stabilizing the protonated Schiff base in the binding pocket.

L3 ANSWER 42 OF 76 MEDLINE on STN DUPLICATE

ACCESSION NUMBER: 2001248737 MEDLINE
DOCUMENT NUMBER: 21214557 PubMed ID: 11313901
TITLE: Rho GTPase-dependent transformation by ***G*** ***protein*** - ***coupled*** ***receptors*** .
AUTHOR: Whitehead I P; Zohn I E; Der C J
CORPORATE SOURCE: Department of Microbiology and Molecular Genetics,
UMDNJ-New Jersey Medical School, Newark, New Jersey, NJ
07103-2714, USA.
CONTRACT NUMBER: CA42978 (NCI)
CA55008 (NCI)
CA63071 (NCI)
CA77493 (NCI)
SOURCE: ONCOGENE, (2001 Mar 26) 20 (13) 1547-55. Ref: 70
Journal code: 8711562. ISSN: 0950-9232.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010517
Last Updated on STN: 20010517
Entered Medline: 20010510

AB ***G*** ***protein*** ***coupled*** ***receptors*** (GPCRs) constitute the largest family of cell surface receptors, with more than 1000 members, and are responsible for converting a diverse array of extracellular stimuli into intracellular signaling events. Most members of the family have defined roles in intermediary metabolism and generally perform these functions in well-differentiated cells. However, there is an increasing awareness that some GPCRs can also regulate proliferative signaling pathways and that chronic stimulation or mutational activation of receptors can lead to oncogenic transformation. Activating mutations in GPCRs are associated with several types of human tumors and some receptors exhibit potent oncogenic activity due to agonist overexpression. Additionally, expression screening analyses for novel oncogenes identified GPCRs whose expression causes the oncogenic transformation of NIH3T3 mouse fibroblasts. These include ***Mas*** , G2A, and the PAR-1 thrombin receptor. In this review we summarize the signaling and transforming properties of these GPCR oncoproteins. What has emerged from these studies is the delineation of a GTPase cascade where transforming GPCRs cause aberrant growth regulation via activation of Rho family small GTPases.

L3 ANSWER 43 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:389863 CAPLUS
DOCUMENT NUMBER: 135:162905
TITLE: Genetic deletion of angiotensin AT2 receptor leads to increased cell numbers in different brain structures of mice
AUTHOR(S): von Bohlen und Halbach, O.; Walther, T.; Bader, M.; Albrecht, D.
CORPORATE SOURCE: Johannes Muller Institute of Physiology (Charite), Humboldt University, Berlin, D-10117, Germany
SOURCE: Regulatory Peptides (2001), 99(2-3), 209-216
CODEN: REPPDY; ISSN: 0167-0115
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Angiotensin II (Ang II) is a potent vasoactive peptide and displays growth factor-like properties. Different high-affinity Ang II receptor subtypes (AT1A, AT1B and AT2) have been cloned. They are expressed in various brain structures. Addnl., it has been assumed that ***Mas*** could interact directly or indirectly with the renin-angiotensin system. The AT1 receptor mediates pressor and mitogenic effects of Ang II, whereas physiol. function and signaling mechanisms of the AT2 receptor remain poorly understood. Recent reports have shown that Ang II could mediate apoptosis through AT2 receptors. Since the AT1A, AT2 and ***Mas*** knockout mice provide new tools for uncovering potential actions of Ang II, the cell no. in different brain structures of male adult wild-type mice and mice deficient for AT1A, AT2 or ***Mas*** was evaluated to get more insight into the role of Ang II in central nervous system development. In nearly all investigated brain structures (cortex, hippocampus, amygdala, thalamus), the cell no. was significantly higher in AT2-deficient mice in comparison to wild-type mice. To the contrary, in AT1A-deficient mice the cell no. was significantly less than in controls in the lateral geniculate and the medial amygdaloid nucleus. However, cell nos. were not changed in ***Mas*** -knockout mice compared to their wild-types. These results show the contrary effects of both angiotensin receptors on cell growth and represent the first demonstration of their action on neuronal cell development evidenced in the adult mouse brain.

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 44 OF 76 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:254773 BIOSIS
DOCUMENT NUMBER: PREV200100254773
TITLE: Molecular biology of light transduction by the mammalian photoreceptor, rhodopsin.
AUTHOR(S): Khorana, Gobind (1)
CORPORATE SOURCE: (1) Massachusetts Institute of Technology, 77 Massachusetts Avenue, Cambridge, MA, 02139 USA
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A167.
print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Rhodopsin, the vertebrate photoreceptor, is a prototypic molecule in the largest family of ***G*** - ***protein*** ***coupled*** ***receptors*** (GPCR). Like all receptors of this family, it contains three distinct domains, the cytoplasmic (intracellular) domain that is involved in all the protein-protein interactions, the transmembrane (TM) domain where the signal transduction begins, by light-catalysed isomerization of 11-cis-retinal to all-trans retinal, and the intradiscal domain which has been shown to be involved in a specific tertiary structure. The main focus of this talk is to describe efforts to understand structures and specific functions of the three domains. The main findings to be presented are as follows: 1. Intradiscal domain contains a globular tertiary structure. A main feature is a disulfide bond (Cys110-Cys187) which is conserved in most of the known GPCR. 2. The correct folding in vivo requires the formation of the above disulfide bond. Misfolding resulting in non-retinal binding is frequently caused by Retinitis Pigmentosa point mutations in the intradiscal and the TM domain. This involves the formation of a C185-C187 disulfide bond. 3. In in vivo folding, the packing of the helices in the TM domain and folding to form the intradiscal tertiary structure are coupled. 4. The first structural event following retinal isomerization is movement or tilting of TM helices as sensed by EPR spectra of spin labeled cysteines placed at selected positions in the cytoplasmic domain. 5. Structural features and light-dependent changes have been studied throughout the cytoplasmic face by systematic cysteine substitutions followed by biochemical studies and EPR spectroscopy. 6. Currently, structure and light-dependent conformational changes are being studied by (a) proximity relationships between different amino acids using disulfide bond formation as the probe; (b) 19F-NMR by attaching trifluoroethyl thio group at different sites on cytoplasmic face through a disulfide bond formation; (c) solution and solid state ***MAS*** NMR by using rhodopsin, 15N-labeled lysine and 15C-labeled glycine and 15N- or 13C-tryptophan.

L3 ANSWER 45 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 18
ACCESSION NUMBER: 2000-572150 [53] WPIDS
DOC. NO. CPI: C2000-170608
TITLE: Determining the existence of a correlation between the pathology of a disease and a gene or mRNA encoding a target polypeptide suspected of being associated with the disease.
DERWENT CLASS: B04 D16
INVENTOR(S): NYCE, J W
PATENT ASSIGNEE(S): (EPIG-N) EPIGENESIS PHARM INC
COUNTRY COUNT: 88
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000051621	A1	20000908	(200053)*	EN	53

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW
AU 2000035123 A 20000921 (200065)
BR 2000009247 A 20011120 (200202)
EP 1165093 A1 20020102 (200209) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
CN 1348376 A 20020508 (200253)
JP 2002537792 W 20021112 (200275) 104
KR 2002068262 A 20020827 (200309)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000051621	A1	WO 2000-US5643	20000302
AU 2000035123	A	AU 2000-35123	20000302
BR 2000009247	A	BR 2000-9247	20000302
		WO 2000-US5643	20000302
EP 1165093	A1	EP 2000-913730	20000302
		WO 2000-US5643	20000302
CN 1348376	A	CN 2000-806759	20000302
JP 2002537792	W	JP 2000-602288	20000302
		WO 2000-US5643	20000302
KR 2002068262	A	KR 2001-711238	20010903

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000035123	A Based on	WO 2000051621
BR 2000009247	A Based on	WO 2000051621
EP 1165093	A1 Based on	WO 2000051621
JP 2002537792	W Based on	WO 2000051621

PRIORITY APPLN. INFO: US 1999-122950P 19990305
AN 2000-572150 [53] WPIDS
AB WO 200051621 A UPAB: 20001023

NOVELTY - A method for determining the existence of a correlation between the pathology of a disease or condition and a gene or mRNA encoding a target polypeptide suspected of being associated with a disease or condition, is new.

DETAILED DESCRIPTION - A method of determining the existence of a correlation between the function of a disease or condition and a gene or mRNA encoding a target polypeptide suspected of being associated with a disease or condition. The method comprises:

(1) obtaining oligonucleotides (oligos) consisting of up to about 15% adenosine (A) and which is anti-sense to a target selected from target genes and their corresponding mRNAs, genomic and mRNA flanking regions selected from 3' and 5' intron-exon borders and the juxta-section between coding and non-coding regions and all mRNA segments encoding polypeptides associated with a pre-selected disease or condition;

(2) selecting an oligo that significantly inhibits or ablates expression of the polypeptide encoded by the mRNA on in vitro hybridization to the target mRNA;

(3) administering the selected oligo to a subject for in vivo hybridization to the target mRNA; and

(4) and assessing the subject's function that is associated with the disease or condition before and after administration of the oligo (a change in the function's value greater than about 70% indicates a positive correlation, about 40-70% a possible correlation and below about 30% a lack of correlation).

USE - The anti-sense oligo is administered to the lung, brain, heart, kidney, tumor, blood, skin, eye, scalp, nose panages, testes, cervix, oral cavity, pharynx, eophagus, small or large intestine, synovial tissue, muscle tissue, ovaries, ear canal or in vitro. The disease or condition afflicts the lung, brain, heart, kidney, tumor, blood, immune system, skin, eye, scalp, nose panages, testes, cervix, oral cavity, pharynx, eophagus, small or large intestine, synovial tissue, muscle tissue, ovaries and ear canal. The disease or condition is particularly one which afflicts the lung (particularly being associated with bronchoconstriction, lung inflammation and/or allergy(ics)), afflicts the brain or is associated with brain activity, is associated with immune dysfunction (particularly where the target is selected from immunoglobulins, antibody receptors, cytokines, cytokine receptors, gene(s) and the corresponding mRNA(s) encoding them, the genes and mRNA flanking regions and intron and exon borders), afflicts the cardiovascular system, associated with the gastrointestinal system or is associated with a malignancy or cancer (particularly where the target is selected from immunoglobulins and antibody receptors, gene(s) and mRNA(s) encoding them, genes and mRNAs associated with oncogenes and genomic and mRNA flanking regions and intron and exon borders). The target gene is selected from genes and mRNAs encoding polypeptides selected from transcription factors, stimulating and activating factors, cytokines and their receptors, interleukins, interleukin receptors, chemokines, chemokine receptors, endogenously produced specific and non-specific enzymes, immunoglobulins, antibod receptors, central nervous system (CNS) and peripheral nervous and non-nervous system receptors, CNS and peripheral nervous and non-nervous system peptide transmitters and their receptors, adhesion molecules, defensines, growth factors, vasoactive peptides and their receptors, peptide receptors and binding proteins and target genes and mRNAs corresponding to oncogenes and their flanking regions and intron and exon borders. The encoded polypeptides are selected from NfkappaB Transcription Factor, Interleukin-8 Receptor (IL-8 R), Interleukin-5 Receptor (IL-5 R), Interleukin-4 Receptor (IL-4 R), Interleukin-3 Receptor (IL-3 R), Interleukin-1beta (IL-1beta), Interleukin-1beta Receptor (IL-1beta R), Eotaxin, Tryptase, Major Basic Protein, beta2-adrenergic Receptor Kinase, Endothelin Receptor A, Endothelin Receptor B, Preproendothelin, Bradykinin B2 Receptor, IgE High Affinity Receptor, Interleukin 1 (IL-1), Interleukin 1 Receptor (IL-1 R), Interleukin 9 (IL-9), Interleukin 9 Receptor (IL-9 R), Interleukin 11 (IL-11), Interleukin 11 Receptor (IL-11 R), Inducible

Nitric Oxide Synthase, Cyclooxygenase (COX), Intracellular Adhesion Molecule 1 (ICAM-1) Vascular Cellular Adhesion Molecule (VCAM), Rantes, Endothelial Leukocyte Adhesion Molecule (ELAM-1), Monocyte Activating Factor, Neutrophil Chemotactic Factor, Neutrophil Elastase, Defensin 1, 2 and 3, Muscarinic Acetylcholine Receptors, Platelet Activating Factor, Tumor Necrosis Factor alpha, 5-lipoxygenase, Phosphodiesterase IV), Substance P, Substance P Receptor, Histamine Receptor, Chymase, CCR-1 CC Chemokine Receptor, CCR-2 CC Chemokine Receptor, CCR-3 CC Chemokine Receptor, CCR-4 CC Chemokine Receptor, CCR-5 CC Chemokine Receptor, Prostanoid Receptors, GATA-3 Transcription Factor, Neutrophil Adherence Receptor, MAP Kinase, Interleukin-9 (IL-9), NFAT Transcription Factors, STAT 4, MIP-1alpha, MCP-2, MCP-3, MCP-4, Cyclophillins, Phospholipase A2, Basic Fibroblast Growth Factor, Metalloproteinase, CSBP/p38 MAP Kinase, Tryptose Receptor, PDG2, Interleukin-3 (IL-3), Interleukin-1beta (IL-1bcta), Cyclosporin A-Binding Protein, FK5-Binding Protein, alpha4eta1 Selectin, Fibronectin, alpha4beta7 Selectin, Mad CAM-1, LFA-1 (CD11a/CD18), PECAM-1, LFA-1, Selectin, C3bi, PSGL-1, E-Selectin, P-Selectin, CD-34, L-Selectin, p150,95, Mac-1 (CD11b/CD18), Fucosyl transferase, VLA-4, CD-18/CD11a, CD11b/CD18, ICAM2 and ICAM3, C5a, CCR3 (Eotaxin Receptor), CCR1, CCR2, CCR4, CCR5, LTB-4, Ap-1 Transcription Factor, Protein kinase C, Cysteinyl Leukotriene Receptor, Tachychinnen Receptors (tach R), IkappaB Kinase 1 and 2, STAT 6, c- ***mas*** and NF-Interleukin-6 (NF-IL-6) and their flanking regions and intron and exon borders.
Dwg.0/4

L3 ANSWER 46 OF 76 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:131474 BIOSIS
DOCUMENT NUMBER: PREV200300131474
TITLE: Molecular biology of light transduction by the mammalian photoreceptor, rhodopsin.
AUTHOR(S): Khorana, H. G. (1)
CORPORATE SOURCE: (1) Departments of Biology and Chemistry, Massachusetts Institute of Technology, 77 Massachusetts Avenue, Bldg. 68-680, Cambridge, MA, 02139, USA: khorana@wccf.mit.edu USA
SOURCE: Sarma, Ramaswamy H. [Editor]; Sarma, Mukti H. [Editor]. (2000) pp. 1-16. Proceedings of the Eleventh Conversation in Biomolecular Stereodynamics. print. Publisher: Adenine Press 2066 Central Avenue, Schenectady, NY, 12304, USA. Meeting Info.: Proceedings of the Eleventh Conversation: Biomolecular Stereodynamics Albany, NY, USA June 15-19, 1999 ISBN: 0-90030-79-9 (cloth), 0-940030-80-2 (cloth), 0-90030-81-0 (cloth).
DOCUMENT TYPE: Book; Conference
LANGUAGE: English

L3 ANSWER 47 OF 76 MEDLINE on STN DUPLICATE 19
ACCESSION NUMBER: 2000387623 MEDLINE
DOCUMENT NUMBER: 20370907 PubMed ID: 10908599
TITLE: A voltage-independent calcium current inhibitory pathway activated by muscarinic agonists in rat sympathetic neurons requires both Galpha q/11 and Gbeta gamma.
AUTHOR: Kammermeier P J; Ruiz-Velasco V; Ikeda S R
CORPORATE SOURCE: Laboratory of Molecular Physiology, Guthrie Research Institute, Sayre, PA 18840, USA.
CONTRACT NUMBER: GM56180 (NIGMS) NS10943 (NINDS) NS37615 (NINDS)
SOURCE: JOURNAL OF NEUROSCIENCE, (2000 Aug 1) 20 (15) 5623-9. Journal code: 8102140. ISSN: 0270-6474.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000818 Last Updated on STN: 20021218 Entered Medline: 20000810
AB Calcium current modulation by the muscarinic cholinergic agonist oxotremorine methiodide (oxo-M) was examined in sympathetic neurons from the superior cervical ganglion of the rat. Oxo-M strongly inhibited calcium currents via voltage-dependent (VD) and voltage-independent (VI) pathways. These pathways could be separated with the use of the specific M(1) acetylcholine receptor antagonist M(1)-toxin and with pertussis toxin (PTX) treatment. Expression by nuclear cDNA injection of the regulator of G-protein signaling (RGS2) or a phospholipase Cbeta1 C-terminal construct (PLCbeta-ct) selectively reduced VI oxo-M modulation in PTX-treated and untreated cells. Expression of the Gbetagamma buffers transducin (Galpha(tr)) and a ***G*** - ***protein*** - ***coupled*** - ***receptor*** kinase (GRK3) construct (***MAS*** -GRK3) eliminated oxo-M modulation. Activation of the heterologously expressed neurokinin type 1 receptor, a Galpha(q/11)-coupled receptor, resulted in VI calcium current modulation. This modulation was eliminated with coexpression of Galpha(tr) or ***MAS*** -GRK3. Cells expressing Gbeta(1)gamma(2) were tonically inhibited via the VD pathway. Application of oxo-M to these cells produced VI modulation and reduced the amount of current inhibited via the VD pathway. Together, these results confirm the requirement for Gbetagamma in VD modulation and implicate Galpha(q)-GTP and Gbetagamma as components in the potentially novel VI pathway.

L3 ANSWER 48 OF 76 MEDLINE on STN DUPLICATE 20
ACCESSION NUMBER: 2001025016 MEDLINE
DOCUMENT NUMBER: 20507688 PubMed ID: 11053263
TITLE: Characterization of gene expression in human trabecular meshwork using single-pass sequencing of 1060 clones.
AUTHOR: Gonzalez P; Epstein D L; Borras T
CORPORATE SOURCE: Department of Ophthalmology, Duke University Medical Center, Durham, North Carolina, USA.
CONTRACT NUMBER: EY01894 (NEI) EY11906 (NEI)
SOURCE: INVESTIGATIVE OPHTHALMOLOGY AND VISUAL SCIENCE, (2000 Nov) 41 (12) 3678-93. Journal code: 7703701. ISSN: 0146-0404.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-BE439390; GENBANK-BE440238
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322 Last Updated on STN: 20010322 Entered Medline: 20001114
AB PURPOSE: To study the gene expression profile of the human trabecular meshwork (HTM). METHODS: A polymerase chain reaction (PCR)-amplified cDNA library was constructed using RNA from the TM of a 67-year-old normal, perfused human eye. A total of 1060 clones were randomly selected for sequencing of one end. These sequences were searched against nonredundant GenBank and dbEST databases for similarity comparison by using a FASTA file and the BLASTcl3 program. Relative expression patterns of those clones that matched other expressed sequence tags (ESTs) were determined using the National Center for Biotechnology Information (NCBI) Unique Human Gene Sequence Collection (UniGene) database. RESULTS: Of the 1060 clones analyzed, 519 (48.9%) had sequences identical with known genes, 125 (11.8%) matched ESTs, and 189 (17.8%) did not match any database sequences. Of the remaining clones, 31 (3%) corresponded to mitochondrial transcripts and 196 (18.5%) to repetitive and noninformative sequences. It is notable that some of the genes highly represented in this library are not ubiquitously expressed in other tissues, which suggests a potentially important role in the HTM. As evidence for the presence of true novel genes in the library, one of the clones was fully sequenced. This clone comprised a complete open reading frame of 966 nucleotides, and its deduced amino acid sequence corresponded to a protein 33% similar to the ***MAS*** -related ***G*** - ***protein*** - ***coupled*** ***receptor*** . CONCLUSIONS: The identification of the more highly expressed genes in HTM and the discovery of novel genes expressed in this tissue provides basic information for further research on the physiology of the TM and for the identification of glaucoma candidate genes.

L3 ANSWER 49 OF 76 MEDLINE on STN DUPLICATE 21
ACCESSION NUMBER: 2000224119 MEDLINE
DOCUMENT NUMBER: 20224119 PubMed ID: 10758111
TITLE: Interaction between ***Mas*** and the angiotensin AT1 receptor in the amygdala.
AUTHOR: Von Bohlen und Halbach O; Walther T; Bader M; Albrecht D
CORPORATE SOURCE: Johannes Muller Institute of Physiology (Charite), Humboldt University, D-10117 Berlin, Germany.
SOURCE: JOURNAL OF NEUROPHYSIOLOGY, (2000 Apr) 83 (4) 2012-21. Journal code: 0375404. ISSN: 0022-3077.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200005
ENTRY DATE: Entered STN: 20000525 Last Updated on STN: 20000525 Entered Medline: 20000515
AB The ***Mas*** -protooncogene is a maternally imprinted gene encoding an orphan ***G*** ***protein*** - ***coupled*** ***receptor*** expressed mainly in limbic structures of the rodent CNS. Because ***Mas*** and the product of the ***Mas*** -related gene enhance the effects of angiotensins on cells expressing angiotensin receptors of the AT1 subtype, we first compared the distribution of cells expressing AT1 receptors in different limbic and thalamic brain structures in ***Mas*** -knockout mice and in wildtype mice by an immunohistochemical approach. No significant differences could be found between the two strains. The ***Mas*** -protooncogene seems to be implicated in the signal transduction of angiotensin receptors and is expressed in the amygdala. Therefore we then analyzed whether field potentials are altered by angiotensin II in brain slices of the basolateral amygdala. An opposite action of angiotensin II was obtained in mice lacking the ***Mas*** -protooncogene in comparison to wildtype mice. The use of different angiotensin receptor antagonists provides the first in vitro evidence for a functional interaction between the ***Mas*** -protooncogene and the AT1 receptor.

L3 ANSWER 50 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2000:448016 CAPLUS
DOCUMENT NUMBER: 133:146458
TITLE: Observations of light-induced structural changes of retinal within rhodopsin
AUTHOR(S): Grobner, Gerhard; Burnett, Ian J.; Glaubitz, Clemens; Choi, Gregory; Mason, A. James; Watts, Anthony
CORPORATE SOURCE: Biomembrane Strticture Unit, Department of Biochemistry, University of Oxford, Oxford, OX1 3QU, UK
SOURCE: Nature (London) (2000), 405(6788), 810-813 CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Photo-isomerization of the 11-cis retinal chromophore activates the mammalian light-receptor rhodopsin, a representative member of a major superfamily of transmembrane ***G*** - ***protein*** - ***coupled*** ***receptor*** proteins (GPCRs) responsible for many cell signal communication pathways. Although low-resoln. (5 A) electron microscopy studies confirm a seven transmembrane helix bundle as a principal structural component of rhodopsin, the structure of the retinal within this helical bundle is not known in detail. Such information is essential for any theor. or functional understanding of one of the fastest occurring photoactivation processes in nature, as well as the general mechanism behind GPCR activation. Here we det. the three-dimensional structure of 11-cis retinal bound to bovine rhodopsin in the ground state at at. level using a new high-resoln. solid-state NMR method. Significant structural changes are obsd. in the retinal following activation by light to the photo-activated MI state of rhodopsin giving the all-trans isomer of the chromophore. These changes are linked directly to the activation of the receptor, providing an insight into the activation mechanism of this class of receptors at a mol. level.
REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 51 OF 76 MEDLINE on STN DUPLICATE 22
ACCESSION NUMBER: 2000149896 MEDLINE
DOCUMENT NUMBER: 20149896 PubMed ID: 10684823
TITLE: Meiosis-activating sterol-mediated resumption of meiosis in mouse oocytes in vitro is influenced by protein synthesis inhibition and cholera toxin.
AUTHOR: Grondahl C; Lessl M; Faerge I; Hegele-Hartung C; Wassermann K; Ottesen J L
CORPORATE SOURCE: Health Care Discovery, Pharmacology, Novo Nordisk A/S, Copenhagen, Denmark. Research Laboratories, Schering AG, Berlin, Germany.. chgr@novo.dk
SOURCE: BIOLOGY OF REPRODUCTION, (2000 Mar) 62 (3) 775-80. Journal code: 0207224. ISSN: 0006-3363.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000421 Last Updated on STN: 20000421 Entered Medline: 20000412
AB To explore the possible signaling pathways of meiosis-activating sterol (***MAS***)-induced oocyte maturation and to elucidate whether the ***MAS*** pathway involves transcription or translation, arrested immature mouse oocytes were cultured with either the protein synthesis

inhibitor cycloheximide or the heteronuclear RNA inhibitors alpha-amanitin
or actinomycin D, respectively. Moreover, the possible involvement of a
G ***protein*** - ***coupled*** ***receptor*** mechanism
in ***MAS*** -mediated oocyte maturation was explored by influencing
oocyte maturation with cholera toxin (CT). ***MAS*** -induced oocyte
maturation was completely blocked by the addition of 50 microg/ml cycloheximide 4 h before the addition of ***MAS*** . Simultaneous addition of ***MAS*** and the protein synthesis inhibitor also significantly reduced the meiotic resumption compared to that in
MAS -treated controls. In contrast, neither of the treatment regimens to inhibit transcription of DNA to RNA was observed to have any
effect on the ***MAS*** -induced resumption of meiosis. CT was observed to inhibit ***MAS*** -induced, but not spontaneous, oocyte
maturation in vitro, suggesting a putative involvement of ***G***
protein - ***coupled*** ***receptor*** mechanism in the
MAS mode of action. In conclusion, protein synthesis was found to
be an essential requirement for maintaining the oocytes' responsiveness to
MAS -induced resumption of meiosis, in contrast to transcription.

L3 ANSWER 52 OF 76 MEDLINE on STN DUPLICATE 23
ACCESSION NUMBER: 2000482717 MEDLINE
DOCUMENT NUMBER: 20428610 PubMed ID: 10971097
TITLE: G protein defects in signal transduction.
AUTHOR: Spiegel A M
CORPORATE SOURCE: National Institute of Diabetes and Digestive and Kidney
Diseases, National Institutes of Health, Bethesda, MD 20892, USA.
SOURCE: HORMONE RESEARCH, (2000) 53 Suppl 3 17-22. Ref: 40
Journal code: 0366126. ISSN: 0301-0163.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20001019
Last Updated on STN: 20001019
Entered Medline: 20001011
AB G proteins couple receptors for many hormones to effectors that regulate
second messenger metabolism. Several endocrine disorders have been shown
to be caused by either loss- or gain-of-function mutations in G proteins
or ***G*** ***protein*** - ***coupled*** ***receptors***

In pseudohypoparathyroidism type Ia (PHP Ia), there are generalized hormone resistance (parathyroid hormone [PTH], thyroid-stimulating hormone, gonadotropins) and associated abnormal physical features, Albright hereditary osteodystrophy. Subjects with PHP Ib are normal in
appearance and show renal resistance to PTH. In McCune-Albright syndrome
(***MAS***), subjects show autonomous endocrine hyperfunction associated with fibrous dysplasia of bone and skin hyperpigmentation.
Germline loss-of-function mutations have been identified in the G(s)-alpha
gene in PHP Ia, and recent evidence suggests that the G(s)-alpha gene is
paternally imprinted in a tissue-specific manner. Abnormal imprinting of
the G(s)-alpha gene may be the cause of PHP Ib. ***MAS*** , in contrast, is caused by gain-of-function missense mutations of the G(s)-alpha gene.
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L3 ANSWER 53 OF 76 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2000:430451 BIOSIS
DOCUMENT NUMBER: PREV200000430451
TITLE: G protein defects in signal transduction.
AUTHOR(S): Spiegel, Allen M. (1)
CORPORATE SOURCE: (1) National Institute of Diabetes and Digestive and Kidney
Diseases, National Institutes of Health, Building 31, Room 9A-52, Bethesda, MD, 20892 USA
SOURCE: Hormone Research (Basel), (August, 2000) Vol. 50, No.
Suppl. 3, pp. 17-22. print.
ISSN: 0301-0163.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB G proteins couple receptors for many hormones to effectors that regulate
second messenger metabolism. Several endocrine disorders have been shown
to be caused by either loss- or gain-of-function mutations in G proteins
or ***G*** ***protein*** - ***coupled*** ***receptors*** . In
pseudohypoparathyroidism type Ia (PHP Ia), there are generalized hormone
resistance (parathyroid hormone (PTH), thyroid-stimulating hormone, gonadotropins) and associated abnormal physical features, Albright hereditary osteodystrophy. Subjects with PHP Ib are normal in
appearance
and show renal resistance to PTH. In McCune-Albright syndrome (***MAS***
), subjects show autonomous endocrine hyperfunction associated with fibrous dysplasia of bone and skin hyperpigmentation. Germline loss-of-function mutations have been identified in the Gs-alpha gene in PHP Ia, and recent evidence suggests that the Gs-alpha gene is paternally
imprinted in a tissue-specific manner. Abnormal imprinting of the Gs-alpha
gene may be the cause of PHP Ib. ***MAS*** , in contrast, is caused by
gain-of-function missense mutations of the Gs-alpha gene.

L3 ANSWER 54 OF 76 MEDLINE on STN DUPLICATE 24
ACCESSION NUMBER: 2000092704 MEDLINE
DOCUMENT NUMBER: 20092704 PubMed ID: 10625868
TITLE: Altered heart rate and blood pressure variability in mice lacking the ***Mas*** protooncogene.
AUTHOR: Walther T; Wessel N; Kang N; Sander A; Tschope C; Malberg
H; Bader M; Voss A
CORPORATE SOURCE: Max-Delbruck-Center for Molecular Medicine, Free University
of Berlin, Berlin, Germany., thowal@mdc-berlin.de
SOURCE: BRAZILIAN JOURNAL OF MEDICAL AND BIOLOGICAL RESEARCH, (2000
Jan) 33 (1) 1-9.
Journal code: 8112917. ISSN: 0100-879X.
PUB. COUNTRY: Brazil
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 20000616
Last Updated on STN: 20000616
Entered Medline: 20000605
AB Heart rate variability is a relevant predictor of cardiovascular risk in humans. A significant genetic influence on heart rate variability is suggested, although the genes involved are ill-defined. The ***Mas***
-protooncogene encodes a ***G*** - ***protein*** - ***coupled***
receptor with seven transmembrane domains highly expressed in
testis and brain. Since this receptor is supposed to interact with the signaling of angiotensin II, which is an important regulator of cardiovascular homeostasis, heart rate and blood pressure were analyzed in
Mas -deficient mice. Using a femoral catheter the blood pressure
of mice was measured for a period of 30 min and 250 data values per second
were recorded. The mean values and range of heart rate and blood pressure
were then calculated. Neither heart rate nor blood pressure were significantly different between knockout mice and controls. However, high
resolution recording of these parameters and analysis of the data by non-linear dynamics revealed significant alterations in cardiovascular variability in ***Mas*** -deficient animals. In particular, females showed a strong reduction of heart rate variability. Furthermore, the data showed an increased sympathetic tone in knockout animals of both genders. The marked alterations detected in ***Mas*** -deficient mice
of both genders suggest that the ***Mas*** -protooncogene is an important determinant of heart rate and blood pressure variability.

L3 ANSWER 55 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN DUPLICATE 25
ACCESSION NUMBER: 2000:894848 SCISEARCH
THE GENUINE ARTICLE: 375PH
TITLE: Molecular biology of light transduction by the mammalian photoreceptor, rhodopsin
AUTHOR: Khorana H G (Reprint)
CORPORATE SOURCE: MIT, DEPT BIOL, BLDG 68-680, 77 MASSACHUSETTS AVE,
CAMBRIDGE, MA 02139 (Reprint); MIT, DEPT CHEM, CAMBRIDGE,
MA 02139
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF BIOMOLECULAR STRUCTURE & DYNAMICS, (NOV 2000)
Sp. iss. S1, pp. 1-16.
Publisher: ADENINE PRESS INC, PO BOX 355/340, GUILDERLAND,
NY 12084.
ISSN: 0739-1102.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 44
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Rhodopsin, the vertebrate photoreceptor, is a prototypic molecule in
the largest family of ***G*** - ***protein*** ***coupled***
receptors (GPCR). Like all receptors of this family, it contains
three distinct domains: the cytoplasmic (intracellular) domain that is involved in all the protein-protein interactions; the transmembrane (TM) domain where the signal transduction begins, by light-catalysed isomerization of 11-cis-retinal to all irans-retinal, and the intradiscal domain which appears to be involved in a specific tertiary structure. The main focus of this talk is to describe efforts to understand specific structure and function in each domain. The main findings to be presented
are as follows: 1. Intradiscal domain contains a globular tertiary structure. A central feature is a disulfide bond (Cys110-Cys187) which is
conserved in most of the known GPCR. 2. The correct folding in vivo requires the formation of the above disulfide bond. Misfolding resulting in non-retinal binding is frequently caused by Retinitis Pigmentosa (RP) point mutations in the intradiscal and the TM domain. 3. In vivo folding studies, using RP mutations in every one of the seven helices, have shown
that the packing of the helices in the TM domain and folding to form the intradiscal tertiary structure are coupled. 4. Cysteine mutagenesis has been used systematically to study the tertiary structure and
light-dependent changes throughout the cytoplasmic face by combination of
biochemical and biophysical studies. In particular, EPR spectroscopy following spin labeling of selected double cysteine mutants has shown movements in helices, including tilting, following retinal isomerization. 5. Large scale expression of mutants has allowed application of both
F-19-NMR (solution) and ***MAS*** solid state NMR tin collaboration
with Dr. Steve Smith's group, SUNY, Stony Brook). Results of current work
are promising for detailed study of the conformational change. Finally, a unifying hypothesis, which is termed the central dogma in the GPCR field,
will be proposed. This states that despite the enormous variation in "accessory" structural details, the principal mechanism of signal transduction starting with pertubation in the seven helical bundle is fundamentally the same in all GPCRs. Experiments to test helix movements,
the first step in signal transduction following ligand binding in two adrenergic receptors are now feasible. The patterns of helix movements in
them will be compared with the pattern demonstrated for rhodopsin and its
mutants.

L3 ANSWER 56 OF 76 MEDLINE on STN DUPLICATE 26
ACCESSION NUMBER: 1999282183 MEDLINE
DOCUMENT NUMBER: 99282183 PubMed ID: 10353830
TITLE: Solid state 15N NMR evidence for a complex Schiff base counterion in the visual ***G*** - ***protein*** - ***coupled*** ***receptor*** rhodopsin.
AUTHOR: Creemers A F; Klaassen C H; Bovee-Geurts P H; Kelle R;
Kragl U; Raap J; de Grip W J; Lugtenburg J; de Groot H J
CORPORATE SOURCE: Leiden Institute of Chemistry, Leiden University, RA
Leiden, The Netherlands.
SOURCE: BIOCHEMISTRY, (1999 Jun 1) 38 (22) 7195-9.
Journal code: 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990712
Last Updated on STN: 20000303
Entered Medline: 19990623
AB Using the baculovirus/Sf9 cell expression system, we have incorporated 99%
15N-enriched [alpha,epsilon-15N2]-L-lysine into the rod visual pigment rhodopsin. We have subsequently investigated the protonated Schiff base
(pSB) linkage in the [alpha, epsilon-15N2]Lys-rhodopsin with cross-polarization magic angle spinning (CP/ ***MAS***) 15N NMR. The
Schiff base (SB) 15N in [alpha, epsilon-15N2]Lys-rhodopsin resonates with
an isotropic shift sigmal of 155.9 ppm, relative to 5.6 M 15NH4Cl. This
suggests that the SB in rhodopsin is protonated and stabilized by a complex counterion. The 15N shifts of retinal SBs correlate with the energy difference between the ground and excited states and the frequency
of maximum visible absorbance, numax, associated with the pi-pi transition
of the polyene chromophore. Experimental modeling of the relation between
the numax and the size of the counterion with a set of pSBs provides strong evidence that the charged chromophore in rhodopsin is stabilized by
a counterion with an estimated effective center-center distance (deff) between the counterion and the pSB of 0.43 +/- 0.01 nm. While selected
prokaryotic proteins and complexes have been labeled before, this is the first time to our knowledge that a 15N-labeled eukaryotic membrane protein
has been generated in sufficient amount for such NMR investigations.

L3 ANSWER 57 OF 76 MEDLINE on STN DUPLICATE 27
ACCESSION NUMBER: 1999110917 MEDLINE
DOCUMENT NUMBER: 99110917 PubMed ID: 9892660
TITLE: Magic angle spinning NMR of the protonated retinylidene Schiff base nitrogen in rhodopsin: expression of 15N-lysine- and 13C-glycine-labeled opsin in a stable cell line.
AUTHOR: Eilers M; Reeves P J; Ying W; Khorana H G; Smith S O
CORPORATE SOURCE: Department of Biochemistry and Cell Biology, State
University of New York, Stony Brook, NY 11794-5215, USA.
CONTRACT NUMBER: GM 28289 (NIGMS)
GM 41412 (NIGMS)
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY

OF SCIENCES OF THE
UNITED STATES OF AMERICA, (1999 Jan 19) 96 (2) 487-92.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990326
Last Updated on STN: 19990326
Entered Medline: 19990316
AB The apoprotein corresponding to the mammalian photoreceptor rhodopsin has
been expressed by using suspension cultures of HEK293S cells in defined
media that contained 6-15N-lysine and 2-13C-glycine. Typical yields were
1.5-1.8 mg/liter. Incorporation of 6-15N-lysine was quantitative, whereas
that of 2-13C-glycine was about 60%. The rhodopsin pigment formed by
binding of 11-cis retinal was spectrally indistinguishable from native
bovine rhodopsin. Magic angle spinning (***MAS***) NMR spectra of
labeled rhodopsin were obtained after its incorporation into liposomes.
The 15N resonance corresponding to the protonated retinylidene Schiff base
nitrogen was observed at 156.8 ppm in the ***MAS*** spectrum of
6-15N-lysine-labeled rhodopsin. This chemical shift corresponds to an
effective Schiff base-counterion distance of greater than 4 A, consistent
with structural water in the binding site hydrogen bonded with the Schiff
base nitrogen and the Glu-113 counterion. The present study demonstrates
that structural studies of rhodopsin and other ***G*** ***protein*** - ***coupled*** ***receptors*** by using ***MAS*** NMR are
feasible.

L3 ANSWER 58 OF 76 MEDLINE on STN DUPLICATE 28
ACCESSION NUMBER: 1999132385 MEDLINE
DOCUMENT NUMBER: 99132385 PubMed ID: 9931487
TITLE: Identification and cloning of three novel human ***G*** ***protein*** - ***coupled*** ***receptor*** genes
GPR52, PsiGPR53 and GPR55: GPR55 is extensively expressed
in human brain.
AUTHOR: Sawzdargo M; Nguyen T; Lee D K; Lynch K R; Cheng R; Heng H
H; George S R; O'Dowd B F
CORPORATE SOURCE: Department of Pharmacology, University of Toronto, Medical
Sciences Building, Toronto, Ontario, Canada, USA.
SOURCE: BRAIN RESEARCH. MOLECULAR BRAIN RESEARCH, (1999 Feb 5) 64
(2) 193-8.
Journal code: 8908640. ISSN: 0169-328X.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF096784; GENBANK-AF096785; GENBANK-AF096786;
GENBANK-AF100789
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990326
Last Updated on STN: 20000303
Entered Medline: 19990312
AB The ***G*** ***protein*** - ***coupled*** ***receptor***
(GPCR) family share a structural motif of seven transmembrane segments
with large numbers of conserved residues in those regions. Here, we
report the identification and cloning of two novel human intronless GPCR
genes, GPR52, GPR55 and a pseudogene PsiGPR53. GPR55 was identified from
the expressed sequence tags (EST) database whereas GPR52 and pseudogene
PsiGPR53 originated from the high throughput genome (HTG) database. A
partial cDNA clone obtained from the IMAGE Consortium of GPR55 was used to
screen a human genomic library to acquire the full length gene. GPR52 and
PsiGPR53 were amplified from human genomic DNA using primers based on the
HTG sequences. GPR55 and GPR52 encode receptors of 319 and 361 amino
acids, respectively. GPR55 gene was mapped to chromosome 2q37, using
fluorescence in situ hybridization (FISH), and its mRNA transcripts have
been detected in the caudate nucleus and putamen, but not in five other
brain regions. Human receptors showing the highest amino acid identity to
GPR55 include P2Y5 (29%), GPR23 (30%), GPR35 (27%) and CCR4 (23%). GPR52
gene localized to chromosome 1q24 shares the highest identity with GPR21
(71%), histamine H2 (27%) and 5-HT4 (26%) human receptors. PsiGPR53 is a
pseudogene mapped to chromosome 6p21 that demonstrates the highest
similarity to the MRG (35%), ***MAS*** (28%) and C5a (24%) human
receptor genes.
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L3 ANSWER 59 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 2000:44079 SCISEARCH
THE GENUINE ARTICLE: 272MQ
TITLE: Thyrotropin-releasing hormone-induced depletion of G(q)alpha/G(11)alpha proteins from detergent-insensitive membrane domains
AUTHOR: Pesanova Z; Novotny J; Cerny J; Milligan G; Svoboda P
(Reprint)
CORPORATE SOURCE: ACAD SCI CZECH REPUBL, INST PHYSIOL, VIDENSKA 1083,
CR-14220 PRAGUE 4, CZECH REPUBLIC (Reprint); ACAD SCI
CZECH REPUBL, INST PHYSIOL, CR-14220 PRAGUE 4, CZECH
REPUBLIC; CHARLES UNIV, FAC NAT SCI, DEPT PHYSIOL & DEV
BIOL, PRAGUE 12800 2, CZECH REPUBLIC; UNIV GLASGOW, INST
BIOMED & LIFE SCI, GLASGOW G12 8QQ, LANARK, SCOTLAND
COUNTRY OF AUTHOR: CZECH REPUBLIC; SCOTLAND
SOURCE: FEBS LETTERS, (24 DEC 1999) Vol. 464, No. 1-2, pp. 35-40.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE
AMSTERDAM, NETHERLANDS.
ISSN: 0014-5793.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 31
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The role of detergent-insensitive membrane domains (DIMs) in desensitisation of the ***G*** ***protein*** - ***coupled*** ***receptor*** -mediated hormone response was studied in clone E2M11 of
HEK293 cells which stably express high levels of both thyrotropin-releasing hormone (TRH) receptors and G(11 alpha) G protein. DIMs were
prepared by flotation in equilibrium sucrose density gradients and characterised by a panel of membrane markers representing peripheral, glycosylphosphatidylinositol-bound as well as integral membrane proteins
(caveolin, CD29, CD55, CD59, CD147, the alpha subunit of Na,K-ATPase) and
enzyme activities (alkaline phosphatase, adenylyl cyclase), Caveolin-containing DIMs represented only a small fraction of the overall
pool of G(q)alpha/G(11)alpha-rich domains. Prolonged stimulation of E2M11
cells with TRH resulted in dramatic depletion of G(q)alpha/G(11)alpha from
all DIMs, which ***mas*** paralleled by a concomitant G(q)alpha/G(11)alpha increase in the high-density gradient fractions containing the bulk-phase membrane constituents soluble in 1% Triton X-100, Distribution of membrane markers ***mas*** unchanged under
these conditions. Membrane domains thus represent a substantial structural
determinant of the G protein pool relevant to desensitisation of hormone
action, (C) 1999 Federation of European Biochemical Societies.

L3 ANSWER 60 OF 76 MEDLINE on STN DUPLICATE 29
ACCESSION NUMBER: 1998234378 MEDLINE
DOCUMENT NUMBER: 98234378 PubMed ID: 9565612
TITLE: Sustained long term potentiation and anxiety in mice lacking the ***Mas*** protooncogene.
AUTHOR: Walther T; Balschun D; Voigt J P; Fink H; Zuschratter W;
Birchmeier C; Ganten D; Bader M
CORPORATE SOURCE: Max-Delbruck-Center for Molecular Medicine (MDC), D-13122
Berlin-Buch, Germany.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 May 8) 273 (19)
11867-73.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980618
Last Updated on STN: 19980618
Entered Medline: 19980605
AB The ***Mas*** protooncogene is a maternally imprinted gene encoding an
orphan ***G*** ***protein*** - ***coupled*** ***receptor***
expressed mainly in forebrain and testis. Here, we provide evidence for a
function of ***Mas*** in the central nervous system. Targeted disruption of the ***Mas*** protooncogene leads to an increased durability of long term potentiation in the dentate gyrus, without affecting hippocampal morphology, basal synaptic transmission, and presynaptic function. In addition, ***Mas*** -/- mice show alterations
in the onset of depotentiation. The permissive influence of ***Mas*** ablation on hippocampal synaptic plasticity is paralleled by behavioral changes. While spatial learning in the Morris water maze is not significantly influenced, ***Mas*** -deficient animals display an increased anxiety as assessed in the elevated-plus maze. Thus, ***Mas*** is an important modulating factor in the electrophysiology of
the hippocampus and is involved in behavioral pathways in the adult brain.

L3 ANSWER 61 OF 76 MEDLINE on STN DUPLICATE 30
ACCESSION NUMBER: 1998147761 MEDLINE
DOCUMENT NUMBER: 98147761 PubMed ID: 9488437
TITLE: ***Mas*** oncogene signaling and transformation require
the small GTP-binding protein Rac.
AUTHOR: Zohn I E; Symons M; Chrzanowska-Wodnicka M; Westwick J K;
Der C J
CORPORATE SOURCE: Department of Pharmacology, Lineberger Comprehensive Cancer
Center, University of North Carolina School of Medicine, Chapel Hill 27599-7038, USA.
CONTRACT NUMBER: CA42978 (NCI)
CA55008 (NCI)
CA63071 (NCI)
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (1998 Mar) 18 (3) 1225-35.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199803
ENTRY DATE: Entered STN: 19980326
Last Updated on STN: 20000303
Entered Medline: 19980319
AB The ***Mas*** oncogene encodes a novel ***G*** - ***protein*** -
coupled ***receptor*** that was identified originally as a transforming protein when overexpressed in NIH 3T3 cells. The mechanism
and signaling pathways that mediate ***Mas*** transformation have not
been determined. We observed that the foci of transformed NIH 3T3 cells
caused by ***Mas*** were similar to those caused by activated Rho and
Rac proteins. Therefore, we determined if ***Mas*** signaling and transformation are mediated through activation of a specific Rho family protein. First, we observed that, like activated Rac1, ***Mas*** cooperated with activated Raf and caused synergistic transformation of NIH
3T3 cells. Second, both ***Mas*** - and Rac1-transformed NIH 3T3 cells
retained actin stress fibers and showed enhanced membrane ruffling.
Third, like Rac, ***Mas*** induced lamellipodium formation in porcine
aortic endothelial cells. Fourth, ***Mas*** and Rac1 strongly activated the JNK and p38, but not ERK, mitogen-activated protein kinases.
Fifth, ***Mas*** and Rac1 stimulated transcription from common DNA
promoter elements: NF-kappaB, serum response factor (SRF), Jun/ATF-2, and
the cyclin D1 promoter. Finally, ***Mas*** transformation and some of
Mas signaling (SRF and cyclin D1 but not NF-kappaB activation)
were blocked by dominant negative Rac1. Taken together, these observations suggest that ***Mas*** transformation is mediated in part
by activation of Rac-dependent signaling pathways. Thus, Rho family proteins are common mediators of transformation by a diverse variety of
oncogene proteins that include Ras, Dbl family, and G-protein-coupled oncogene proteins.

L3 ANSWER 62 OF 76 MEDLINE on STN DUPLICATE 31
ACCESSION NUMBER: 1998355528 MEDLINE
DOCUMENT NUMBER: 98355528 PubMed ID: 9692779
TITLE: Mutational analysis of the potential phosphorylation sites for protein kinase C on the CCK(A) receptor.
AUTHOR: Smeets R L; Fouraux M A; Pouwels W; van Emst-de Vries S E;
Ronken E; De Pont J J; Willems P H
CORPORATE SOURCE: Department of Biochemistry, University of Nijmegen, The
Netherlands.
SOURCE: BRITISH JOURNAL OF PHARMACOLOGY, (1998 Jul) 124 (5) 935-45.
Journal code: 7502536. ISSN: 0007-1188.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 19981020
Last Updated on STN: 20000303
Entered Medline: 19981002
AB 1. Many ***G*** ***protein*** - ***coupled*** ***receptors***
contain potential phosphorylation sites for protein kinase C (PKC), the exact role of which is poorly understood. In the present study, a mutant cholecystokininA (CCK(A)) receptor was generated in which the four consensus sites for PKC action were changed in an alanine. Both the wild-type (CCK(A)WT) and mutant (CCK(A)MT) receptor were stably

expressed
in Chinese hamster ovary (CHO) cells. 2. Binding of [3H]-cholecystokinin-
(26-33)-peptide amide (CCK-8) to membranes prepared from CHO-CCK(A)WT
cells and CHO-CCK(A)MT cells revealed no difference in binding affinity
(Kd values of 0.72 nM and 0.86 nM CCK-8, respectively). 3. The dose-response curves for CCK-8-induced cyclic AMP accumulation and inositol 1,4,5-trisphosphate (Ins(1,4,5)P3) formation were shifted to the left in CHO-CCK(A)MT cells. This leftward shift was mimicked by the potent inhibitor of protein kinase activity, staurosporine. However, the effect of staurosporine was restricted to CHO-CCK(A)WT cells. This demonstrates that attenuation of CCK-8-induced activation of adenylyl cyclase and phospholipase C-beta involves a staurosporine-sensitive kinase, which acts directly at the potential sites of PKC action on the CCK(A) receptor in CCK-8-stimulated CHO-CCK(A)WT cells. 4. The potent PKC
activator, 12-O-tetradecanoylphorbol 13-acetate (TPA), evoked a rightward
shift of the dose-response curve for CCK-8-induced cyclic AMP accumulation
in CHO-CCK(A)WT cells but not CHO-CCK(A)MT cells. This is in agreement
with the idea that PKC acts directly at the CCK(A) receptor to attenuate
adenylyl cyclase activation. 5. In contrast, TPA evoked a rightward shift
of the dose-response curve for CCK-8-induced Ins(1,4,5)P3 formation in
both cell lines. This demonstrates that high-level PKC activation inhibits CCK-8-induced Ins(1,4,5)P3 formation also at a post-receptor site. 6. TPA inhibition of agonist-induced Ca2+ mobilization was only partly reversed in CHO-CCK(A)MT cells. TPA also inhibited Ca2+ mobilization in response to the G protein activator, ***Mas*** -7. These findings are in agreement with the idea that partial reversal of agonist-induced Ca2+ mobilization is due to the presence of an additional
site of PKC inhibition downstream of the receptor and that the mutant receptor itself is not inhibited by the action of PKC. 7. The data presented demonstrate that the predicted sites for PKC action on the CCK(A) receptor are the only sites involved in TPA-induced uncoupling of
the receptor from its G proteins. In addition, the present study unveils a post-receptor site of PKC action, the physiological relevance of which may be that it provides a means for the cell to inhibit phospholipase C-beta activation by receptors that are not phosphorylated by PKC.

L3 ANSWER 63 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 1999:43393 SCISEARCH
THE GENUINE ARTICLE: 152PE
TITLE: Polarized cell surface expression of the green fluorescent protein-tagged vasopressin V2 receptor in Madin Darby canine kidney cells
AUTHOR: Schulein R (Reprint); Lorenz D; Oksche A; Weisner B; Hermosilla R; Ebert J; Rosenthal W
CORPORATE SOURCE: FORSCHUNGSINST MOL PHARMAKOL, ALFRED KOWALKE STR 4,
D-10315 BERLIN, GERMANY (Reprint); UNIV GIESSEN, RUDOLF
BUCHHEIM INST PHARMAKOL, D-35392 GIESSEN, GERMANY; FREE
UNIV BERLIN, INST PHARMAKOL, D-14195 BERLIN, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: FEBS LETTERS, (18 DEC 1998) Vol. 441, No. 2, pp. 170-176.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE
AMSTERDAM, NETHERLANDS.
ISSN: 0014-5793.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 18
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB We have analyzed the polarized cell surface expression of the G protein-coupled vasopressin V2 receptor (V2 receptor) in Madin-Darby canine kidney (MDCK) epithelial cells by both conventional cell surface biotinylation assays and laser scanning microscopy of green fluorescent protein (GFP)-tagged receptors, Cell surface biotinylation assays with stably transfected filter-grown cells expressing alkaline phosphatase (PhoA)-tagged receptors demonstrated that the V2 receptor is located predominantly basolaterally at steady state, while minor amounts are expressed apically. Laser scanning microscopy of filter- and glass-grown
MDCK cells stably transfected with a GFP-tagged V2 receptor confirmed that
the receptor is expressed mainly basolaterally; within the basolateral compartment, however, the receptor ***mas*** confined to the lateral
subdomain, The results obtained with the GFP-tagged receptor are thus consistent with and refine those from the biotinylation assay, which does not discriminate lateral from basal membrane regions. Our data indicate that the GFP methodology may effectively supplement cell surface biotinylation assays in future studies of polarized receptor transport. We finally show that microinjection of a plasmid encoding the GFP-tagged V2
receptor into the nucleus of MDCK cells led to the same results as experiments with stably transfected cells. However, since there was no need for selecting stably transfected cell lines, the experiments were complete within hours. The microinjection technique thus constitutes a powerful single cell technique to study the intracellular transport of ***G*** ***protein*** - ***coupled*** ***receptors*** , The
methodology may be applicable to any cell type, even to tissue-derived, primary cultured cells; coinjection of transport-regulating compounds should also be possible, (C) 1998 Federation of European Biochemical Societies.

L3 ANSWER 64 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2000:256286 CAPLUS
DOCUMENT NUMBER: 133:26902
TITLE: Angiotensin II receptors AT1 and AT2: new mechanisms
of signaling and antagonistic effects of AT1 and AT2
AUTHOR(S): Inagami, Tadashi; Eguchi, Satoru; Tsuzuki, Satoshi;
Ichiki, Toshihiro
CORPORATE SOURCE: Vanderbilt University School of Medicine, Nashville,
TN, 37232-0146, USA
SOURCE: Progress in Experimental Cardiology (1998), 2(Angiotensin II Receptor Blockade: Physiological and Clinical Implications), 129-139
CODEN: PEXCFF; ISSN: 1389-1774
PUBLISHER: Kluwer Academic Publishers
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review, with 139 refs. A series of pharmaceutical successes in the treatment of not only essential hypertension but also vascular hypertrophic and hyperplastic diseases, congestive heart failure, and renal degenerative diseases, with angiotensin-converting enzyme inhibitors
and angiotensin (Ang) II receptor antagonists indicates that angiotensin may play a pivotal role in the genesis and maintenance of high blood pressure and resultant stroke, atherosclerosis, and heart and kidney diseases. There is more than one form of Ang II receptors. Using expression cloning, the authors isolated the AT1 cDNA from bovine adrenocortical cells from the kidney of spontaneously hypertensive rats and AT2 cDNA from rat PC12W cells and the authors showed that it was not
the ***mas*** oncogene product. Further, the authors showed that in
rodents, AT1 consists of two subtypes, AT1A and AT1B, which share a high
degree of sequence homol. in their coding regions, although mechanisms of
their resp. transcriptional control seemed to be different. By computer-assisted modeling and site-directed mutagenesis, the authors have
delineated the docking site of Ang II. AT1a (and AT1b) serves most of the
commonly recognized actions of Ang II. In addn., this ***G*** ***protein*** - ***coupled*** ***receptor*** (GPCR) also activates
a tyrosine kinase mechanism that may be an underlying cause of Ang II-mediated hypertrophic and hyperplastic changes of cardiovascular tissues. In the vascular system, the phospholipase C (PLC) activated by Ang II seems to be PLC-.beta. rather than PLC-.gamma.1. Interestingly,
the authors found that Gq-activated PLC-.beta. activates p21 ras and mitogen-activated protein kinase (MAPK) in rat vascular smooth muscle
cells. The mechanism of the cross-talk between AT1 and the tyrosine kinase system is triggered by Ca2+, but does not involve protein kinase C.
Studies using targeted gene deletion indicated that Ang II is intimately involved in nephrogenesis. Mice lacking angiotensinogen showed an abnormality in the formation of renal papilla, retardation in glomerular maturation, marked hypertrophy of small arteries of the kidney, and tubular dilatation, whereas targeted deletion of the AT1 receptor resulted
in small arterial wall hypertrophy. Blood pressure of AT1A-deleted mice
was markedly reduced (-45 mmHg). The role and mechanism of action of AT2
was not clear. The authors have recently produced AT2 gene null mice and
AT1a knockout mice by targeted gene deletion. AT2-deleted mice had a
higher blood pressure, whereas AT1-deleted mice showed lower blood pressure. Deletion of the AT2 gene also showed reduced exploratory activity. The most conspicuous action of the AT2 receptor is seen in its salt-retaining action in the renal tubule. Under a const. renal blood flow condition an AT2 antagonist markedly increased the urine vol. and concomitant natriuresis. These effects are completely abolished in AT2 deleted mice. The mol. and cell biol. studies of the angiotensin receptors are needed. Despite the complexity and often mutually antagonistic actions of AT1 and AT2, Ang II, working through AT1 and AT2
of the kidney work in the same direction to retain salt and water. These observations, as well as the effects of Ang II, indicate that the most fundamental role of Ang II is its role in the development of the salt-retaining organ, the kidney, and Ang II is uniquely related to the kidney in that both AT1 and AT2 receptors work for the retention of salt.
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 65 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 1998:941233 SCISEARCH
THE GENUINE ARTICLE: 145AK
TITLE: Characterization of the 5'-flanking promoter region of the rat somatostatin receptor subtype 3 gene
AUTHOR: Glos M; Kreienkamp H J; Hausmann H; Richter D (Reprint)
CORPORATE SOURCE: UNIV HAMBURG, UKE, INST ZELLBIOCHEM & KLIN NEUROBIOL,
MARTINISTR 52, D-20246 HAMBURG, GERMANY (Reprint); UNIV
HAMBURG, UKE, INST ZELLBIOCHEM & KLIN NEUROBIOL, D-20246
HAMBURG, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: FEBS LETTERS, (27 NOV 1998) Vol. 440, No. 1-2, pp. 33-37.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE
AMSTERDAM, NETHERLANDS.
ISSN: 0014-5793.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 16
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB We investigated the 5'-flanking promoter region of the rat somatostatin
receptor subtype 3 (rSSTR3), Using a cDNA probe, genomic clones containing
the 5'-flanking promoter region of the rSSTR3 gene were isolated. A sequence of 5.4 kb directly upstream from the start codon ***mas*** analyzed and two introns mere found in the 5' untranslated region (UTR) of
the cDNA sequence, The transcriptional initiation site was determined by
5' rapid amplification of cDNA ends (RACE), primer extension and RNase
protection analysis with cerebellar RNA. Two major transcriptional initiation sites were found at position -1040 (tsp1) and -856 (tsp2) relative to the translational initiation site. Like a number of other promoters of ***G*** - ***protein*** - ***coupled*** ***receptors*** , the rSSTR3 gene lacks TATA and CAAT motifs and includes
G+C-rich regions. Functional analysis of the promoter region by transfecting rSSTR3 luciferase-reporter gene constructs into rat pituitary
GH(3) cells and HEK 293 cells indicated that a 107-bp region upstream of
tsp2 was sufficient to drive transcription. Furthermore a 562-bp region at
position -1304 to -1865 upstream of the ATG start codon exerted a negative
regulatory effect on transcriptional activity. (C) 1998 Federation of European Biochemical Societies.

L3 ANSWER 66 OF 76 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1997:315326 CAPLUS
DOCUMENT NUMBER: 126:289718
TITLE: Analysis of receptor function in G protein-coupled signal transduction using the yeast mating factor system
INVENTOR(S): Das, Prem O.; Mandell, Robert B.; Boulton, Teri G.;
McMullen, Thomas W.
PATENT ASSIGNEE(S): Heartland Biotechnologies, L.L.C., USA; Das, Prem O.;
Mandell, Robert B.; Boulton, Teri G.; McMullen, Thomas W.
SOURCE: PCT Int. Appl., 108 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9711159	A1	19970327	WO 1996-US15203	19960920
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2232733	AA	19970327	CA 1996-2232733	19960920
AU 9671158	A1	19970409	AU 1996-71158	19960920
EP 862614	A1	19980909	EP 1996-932305	19960920
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1995-4023P P 19950920
WO 1996-US15203 W 19960920
AB A method of analyzing G protein-coupled signal transduction using fusion
proteins of the .alpha. subunit of the yeast G protein involved in the yeast pheromone system is described. The N-terminal moiety of the fusion
protein is a ***G*** ***protein*** - ***coupled*** ***receptor*** and the C-terminal moiety is the G protein .alpha. subunit. The coupling between the two proteins allows the use of the system to assay and identify ligands for the receptor even when its endogenous ligand is unknown (an orphan receptor.). Methods are described
for creating DNA constructs that encode such fusion protein, assays for correct expression of such fusion mols. in yeast, and assays for the coupling of such fusion mols. to the pheromone-induced signal transduction
pathway of yeast. The construction of hosts for the expression of these constructs is also described. Furthermore, the invention encompasses yeasts expressing the fusion proteins and methods for screening compds.
for activity as agonists or antagonists of seven-transmembrane receptor function. Thrombin activation of the yeast pheromone signal transduction
pathway through a fusion protein of the thrombin receptor and gene GPA1 G
protein is reported.

L3 ANSWER 67 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN

ACCESSION NUMBER: 97:607763 SCISEARCH
THE GENUINE ARTICLE: XQ124
TITLE: Direct interaction of G beta gamma with a C-terminal G beta gamma-binding domain of the Ca2+ channel alpha(1) subunit is responsible for channel inhibition by ***G*** ***protein*** - ***coupled*** ***receptors***
AUTHOR: Qin N (Reprint); Platano D; Olcese R; Stefani E; Birnbaumer L
CORPORATE SOURCE: UNIV CALIF LOS ANGELES, DEPT ANESTHESIOL, LOS ANGELES, CA 90095 (Reprint); UNIV CALIF LOS ANGELES, DEPT BIOL CHEM, LOS ANGELES, CA 90095; UNIV CALIF LOS ANGELES, DEPT PHYSIOL, LOS ANGELES, CA 90095; UNIV CALIF LOS ANGELES, DEPT BRAIN RES, LOS ANGELES, CA 90095; UNIV CALIF LOS ANGELES, MOL BIOL RES INST, LOS ANGELES, CA 90095
COUNTRY OF AUTHOR: USA
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (5 AUG 1997) Vol. 94, No. 16, pp. 8866-8871.
Publisher: NATL ACAD SCIENCES, 2101 CONSTITUTION AVE NW, WASHINGTON, DC 20418.
ISSN: 0027-8424.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 50
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Several classes of voltage-gated Ca2+ channels (VGCCs) are inhibited by G proteins activated by receptors for neurotransmitters and neuromodulatory peptides, Evidence has accumulated to indicate that for non-L-type Ca2+ channels the executing arm of the activated G protein is its beta gamma dimer (G beta gamma). We report below the existence of two G beta gamma-binding sites on the A-, B-, and E-type alpha(1) subunits that form non-L-type Ca2+ channels. One, reported previously, is in loop 1 connecting transmembrane domains I and II, The second is located approximately in the middle of the ca, 600-aa-long C-terminal tails, Both G beta gamma-binding regions also bind the Ca2+ channel beta subunit (CC beta), which, when overexpressed, interferes with inhibition by activated G proteins, Replacement in alpha(1E) Of loop 1 with that of the G protein-insensitive and G beta gamma-binding-negative loop 1 of alpha(1C) did not abolish inhibition by G proteins, but the exchange of the alpha(1E) C terminus with that of alpha(1C) did, This and properties of alpha(1E) C-terminal truncations indicated that the G beta gamma-binding site mediating the inhibition of Ca2+ channel activity is the one in the C terminus, Binding of G beta gamma to this site ***mas*** inhibited by an alpha(1)-binding domain of CC beta, thus providing an explanation for the functional antagonism existing between CC beta and G protein inhibition. The data do not support proposals that G beta gamma inhibits alpha(1) function by interacting with the site located in the loop I-II linker, These results define the molecular mechanism by which presynaptic ***G*** ***protein*** - ***coupled*** ***receptors*** inhibit neurotransmission.

L3 ANSWER 68 OF 76 MEDLINE on STN DUPLICATE 32
ACCESSION NUMBER: 2000409241 MEDLINE
DOCUMENT NUMBER: 20362178 PubMed ID: 10904553
TITLE: Spin-labeled extracellular loop from a seven-transmembrane helix receptor: studies in solution and interaction with model membranes.
AUTHOR: Pertinhez T A; Nakaie C R; Paiva A C; Schreier S
CORPORATE SOURCE: Departamento de Bioquimica, Universidade de Sao Paulo, Brazil.
SOURCE: BIOPOLYMERS, (1997 Dec) 42 (7) 821-9.
Journal code: 0372525. ISSN: 0006-3525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000907
Last Updated on STN: 20000907
Entered Medline: 20000828
AB A spin-labeled pentadecapeptide was synthesized containing 2,2,6,6-tetramethylpiperidine-N-oxyl-4-amino-4-carboxylic acid (TOAC) as the N-terminal amino acid and residues 253-266 (EYWSTFGNLHHISL) of the mass oncogene receptor, a membrane-bound protein from the ***G*** - ***protein*** ***coupled*** ***receptors*** family. According to predictions, this protein folds into seven transmembrane helices connected by three extra- and three intracellular loops, and the peptide encompasses part of the third extracellular loop and part of the seventh helix. Electron paramagnetic resonance (EPR) spectra of the spin-labeled peptide (TOAC-14) were obtained in aqueous solution as a function of pH and temperature, in a secondary structure-inducing solvent [trifluoroethanol (TFE)], and in the presence of detergent micelles and phospholipid bilayers. The charged and uncharged amino groups of TOAC and TOAC-14 yielded spectra with different isotropic hyperfine splittings (aN). The slow exchange between protonated and unprotonated forms in the EPR time scale gave rise to composite spectra weighted by the Henderson-Hasselbalch equation. Plots of aN vs pH allowed the determination of the amino group pK values (8.4 and 4.5, for TOAC and TOAC-14, respectively). A small change in aN centered at pH 6.5 was ascribed to the titration of the histidines. Values of calculated rotational correlation times were indicative of a pH-induced conformational change. A conformational change was also observed in TFE. TOAC-14 bound to micelles irrespective of peptide and detergent head group charge. In contrast, the peptide bound to phospholipid bilayers only when both carried opposite charges. The slow exchange (in the EPR time scale) between membrane-bound and free TOAC-14 allowed the calculation of the peptide's partition coefficient. The spectral line shapes were affected by aggregate size and degree of packing of the constituent molecules. It is proposed that pH, polarity, and lipid environment can affect the conformation of water-exposed regions of membrane-bound receptors, thereby playing a role in the mechanism of signal transduction.

L3 ANSWER 69 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 97:135175 SCISEARCH
THE GENUINE ARTICLE: WG234
TITLE: The activation process of the alpha(1B)-adrenergic receptor: Potential role of protonation and hydrophobicity of a highly conserved aspartate
AUTHOR: Scheer A; Fanelli F; Costa T; DeBenedetti P G; Cotecchia S (Reprint)
CORPORATE SOURCE: UNIV LAUSANNE, INST PHARMACOL & TOXICOL, CH-1005 LAUSANNE, SWITZERLAND (Reprint); UNIV LAUSANNE, INST PHARMACOL & TOXICOL, CH-1005 LAUSANNE, SWITZERLAND; UNIV MODENA, DIPARTIMENTO CHIM, I-41100 MODENA, ITALY; IST SUPER SANITA, I-00161 ROME, ITALY
COUNTRY OF AUTHOR: SWITZERLAND; ITALY
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (4 FEB 1997) Vol. 94, No. 3, pp. 808-813.
Publisher: NATL ACAD SCIENCES, 2101 CONSTITUTION AVE NW, WASHINGTON, DC 20418.
ISSN: 0027-8424.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 30
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB In this study, a quantitative approach ***mas*** used to investigate the role of D142, which belongs to the highly conserved E/DRY sequence, in the activation process of the alpha(1B)-adrenergic receptor (alpha(1B)-AR). Experimental and computer-simulated mutagenesis mere performed by substituting all possible natural amino acids at the D142 site. The resulting congeneric set of proteins together with the finding that all the receptor mutants show various levels of constitutive (agonist-independent) activity enabled us to quantitatively analyze the relationships between structural/dynamic features and the extent of constitutive activity. Our results suggest that the hydrophobic/hydrophilic character of D142, which could be regulated by protonation/deprotonation of this residue, is an important modulator of the transition between the inactive (R) and active (R*) state of the alpha(1B)-AR. Our study represents an example of quantitative structure-activity relationship analysis of the activation process of a ***G*** ***protein*** - ***coupled*** ***receptor*** .

L3 ANSWER 70 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 96:80956 SCISEARCH
THE GENUINE ARTICLE: TP937
TITLE: P-2 PURINOCEPTOR-MEDIATED DEPOLARIZATION OF RAT SUPRAOPTIC NEUROSECRETORY-CELLS IN-VITRO
AUTHOR: HIRUMA H; BOURQUE C W (Reprint)
CORPORATE SOURCE: MONTREAL GEN HOSP, DIV NEUROL, CTR RES NEUROSCI, 1650 CEDAR AVE, MONTREAL, PQ H3G 1A4, CANADA (Reprint); MONTREAL GEN HOSP, DIV NEUROL, CTR RES NEUROSCI, MONTREAL, PQ H3G 1A4, CANADA; MCGILL UNIV, MONTREAL, CANADA
COUNTRY OF AUTHOR: CANADA
SOURCE: JOURNAL OF PHYSIOLOGY-LONDON, (15 DEC 1995) Vol. 489, No. 3, pp. 805-811.
ISSN: 0022-3751.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 21
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB 1. Intracellular recordings were obtained from supraoptic magnocellular neurosecretory cells (MNCs) in superfused explants of rat hypothalamus. Application of ATP and UTP, but not adenosine, produced TTX-insensitive depolarizations accompanied by increases of input conductance. 2. The P-2X agonists alpha,beta-methylene ATP, beta,gamma-methylene ATP and 2-methylthio ATP mimicked the effects of ATP in >77% of the cells tested. Depolarizing responses to ATP were reversibly inhibited by PPADS (pyridoxal-phosphate-6-azophenyl-2',4'-disulphonic acid; IC50 approximate to 0.5 mu M), a selective P-2X antagonist. 3. The reversal potential. of responses to ATP (-37 mV) was not strongly affected by intracellular Cl- injection or by removal of Cl- from the external solution. The reversal potential of responses to the most potent P-2X agonist, alpha,beta-methylene ATP, ***mas*** -29 mV. These values suggest the involvement of non-selective cationic channels, a finding which is consistent with the ionotropic cationic channel structure of cloned P-2X purinoceptors. 4. The reversal potential of UTP-mediated responses (-33 mV) was also consistent with the involvement of non-selective cationic channels. Since cloned P-2U receptors display homology with ***G*** - ***protein*** - ***coupled*** ***receptors*** , cationic channels modulated by UTP are probably different from those mediating P-2X responses.

L3 ANSWER 71 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 95:552890 SCISEARCH
THE GENUINE ARTICLE: RP176
TITLE: CANNABINOIDS ENHANCE HUMAN B-CELL GROWTH AT LOW NANOMOLAR CONCENTRATIONS
AUTHOR: DEROCQ J M (Reprint); SEGUI M; MARCHAND J; LEFUR G; CASELLAS P
CORPORATE SOURCE: SANOFI RECH, DEPT IMMUNOL, 371 RUE PROFESSEUR BLAYAC, F-34184 MONTPELLIER 04, FRANCE (Reprint)
COUNTRY OF AUTHOR: FRANCE
SOURCE: FEBS LETTERS, (07 AUG 1995) Vol. 369, No. 2-3, pp. 177-182
ISSN: 0014-5793.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 25
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB This study examined the effect of cannabinoid ligands on human tonsillar B-cells activated either through cross-linking of surface immunoglobulins or ligation of the CD40 antigen. The two synthetic cannabinoids, CP55,940 and WIN55212-2, as well as Delta(9)-tetrahydrocannabinol (THC), the psychoactive component of marijuana, caused a dose-dependent increase of B-cell proliferation and displayed EC(50) at low nanomolar concentrations. This cannabinoid-induced enhancing activity ***mas*** inhibited by pertussis toxin which suggested a ***G*** - ***protein*** - ***coupled*** ***receptor*** process. In addition, the absence of antagonistic effect of SR141716A, a specific CB1 receptor antagonist, together with the demonstration that human B-cells displayed large amount of CB2 receptor mRNAs, led us to assume that the growth enhancing activity observed on B-cells at very low concentrations of cannabinoids could be mediated through the CB2 receptor.

L3 ANSWER 72 OF 76 SCISEARCH COPYRIGHT 2003 THOMSON ISI on STN
ACCESSION NUMBER: 95:40677 SCISEARCH
THE GENUINE ARTICLE: PZ573
TITLE: EXPRESSION OF THE MOUSE AND RAT ***MAS*** PROTOONCOGENE IN THE BRAIN AND PERIPHERAL-TISSUES
AUTHOR: METZGER R; BADER M (Reprint); LUDWIG T; BERBERICH C; BUNNEMANN B; GANTEN D
CORPORATE SOURCE: MAX DELBRUCK CTR MOLEC MED, HYPERTENS RES, BLDG 134D, WILTBERGSTR 50, D-13125 BERLIN, GERMANY (Reprint); MAX DELBRUCK CTR MOLEC MED, HYPERTENS RES, D-13125 BERLIN,

GERMANY; EUROPEAN MOLEC BIOL LAB, HEIDELBERG, GERMANY;
GERMAN INST HIGH BLOOD PRESSURE RES, D-69120 HEIDELBERG, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: FEBS LETTERS, (02 JAN 1995) Vol. 357, No. 1, pp. 27-32.
ISSN: 0014-5793.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 46
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We isolated the mns proto-oncogene from a mouse genomic library, Sequence analysis showed that it contains an open reading frame without intervening sequences, The amino acid sequence deduced confirms the seven-transmembrane-domain structure and exhibits 97% and 91% amino acid homology with the rat and the human ***Mas*** , respectively, In mice and rats, inns mRNA was detected in the testis, kidney, heart, and in the brain regions: hippocampus, forebrain, piriform cortex, and olfactory bulb, Testicular ***mas*** mRNA from rats increases markedly during development, while cerebellar mRNA is high postnatally but completely disappears at later stages, We conclude that the product of the mouse ***mas*** gene may be involved in the development of the brain and testis.

L3 ANSWER 73 OF 76 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN DUPLICATE 33
ACCESSION NUMBER: 1994-101120 [12] WPIDS
CROSS REFERENCE: 1996-208785 [21]
DOC. NO. CPI: C1994-046615
TITLE: Polypeptides of G-coupled receptor proteins (GPRs) - useful for binding GPR ligands or modulating GPR binding.
DERWENT CLASS: B04 D16
INVENTOR(S): MURPHY, R B; SCHUSTER, D I
PATENT ASSIGNEE(S): (UYNY) UNIV NEW YORK STATE
COUNTRY COUNT: 19
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9405695	A1	19940317	(199412)*	EN	160
RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE					
W: AU CA JP					
AU 9348553	A	19940329	(199430)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9405695	A1	WO 1993-US8528	19930909
AU 9348553	A	AU 1993-48553	19930909

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 9348553	A Based on	WO 9405695

PRIORITY APPLN. INFO: US 1992-943236 19920910
AN 1994-101120 [12] WPIDS
CR 1996-208785 [21]
AB WO 9405695 A UPAB: 19960604
A ***G*** - ***protein*** ***coupled*** ***receptor*** polypeptide (A) comprises 15-40 amino acids corresp. to a fragment or consensus peptide of a transmembrane domain of a ***G*** - ***protein*** ***coupled*** ***receptor*** (GPR) and has biological activity selected from binding a GPR ligand or modulating GPR binding to a GPR.

Also claimed are: (1) an antibody (Ab), anti-idiotype Ab or fragments, specifically displaying an epitope of (A); (2) the nucleic acid (I) encoding (A); (3) a vector comprising (I); and (4) a host cell comprising (I).

(A) has a sequence selected from those given in the specification, e.g. DDIFVTLDVLFSTASILNLSAISLKKK.

The GPR is selected from a CAMP receptor, adenosine receptor, beta-adrenergic receptor, muscarinic acetylcholine receptor, alpha-adrenergic receptor, serotonin receptor, histamine H2 receptor, thrombin receptor, Kinin receptor, follical stimulating hormone (FSH) receptor, opsin, rhodopsin, odorant receptor, CMV receptor or ***mas*** oncogene GPR. The transmembrane domain of (A) is selected from TM1, TM2, TM3, TM4, TM5, TM6, TM7 or D2 receptor transmembrane segment III or V. The compsn. further comprises a drug selected from a phenothiazine deriv., thioxanthine deriv., butyrophenone deriv., dihydroindolone, dibenzoxozepine deriv., or atypical neuroleptic.

USE - (A) is useful for binding GPR ligands or modulating GPR ligand binding to GPR. (A) is useful in a compns. (claimed) for treating subjects suffering from a pathology related to a GPR abnormality, e.g. a psychotic disorder such as schizophrenia. It can be used to isolate a GPR fragment or consensus sequence or a protein binding the GPR.(A) is administered by oral, mucosal, intravenous, intramuscular or parenteral route in an amt. ranging from 0.01 micro.g-100 mg/kg/day, esp. 10 micro.g-10 mg/kg/day.
Dwg.0/8
Dwg.0/8

L3 ANSWER 74 OF 76 MEDLINE on STN DUPLICATE 34
ACCESSION NUMBER: 94049026 MEDLINE
DOCUMENT NUMBER: 94049026 PubMed ID: 8231733
TITLE: Expression of the ***mas*** proto-oncogene in the rat hippocampal formation is regulated by neuronal activity.
AUTHOR: Martin K A; Hockfield S
CORPORATE SOURCE: Section of Neurobiology, Yale University School of Medicine, New Haven, CT 06510.
CONTRACT NUMBER: NS22807 (NINDS)
SOURCE: BRAIN RESEARCH. MOLECULAR BRAIN RESEARCH, (1993 Sep) 19 (4) 303-9.
Journal code: 8908640. ISSN: 0169-328X.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199312
ENTRY DATE: Entered STN: 19940117
Last Updated on STN: 19970203
Entered Medline: 19931201

AB The ***mas*** proto-oncogene encodes a seven membrane-spanning ***G*** - ***protein*** - ***coupled*** ***receptor*** which is activated by angiotensins. In the postnatal and adult rat, ***mas*** mRNA is specifically expressed at high levels in hippocampal neurons. We report here using in situ hybridization and RNase protection that brief seizure episodes lead to a significant and transient increase in ***mas*** mRNA in the hippocampus. Increased levels of ***mas*** transcripts were detected 2, 4, and 6 h following seizure. By 24 h post seizure, baseline levels were detected. The presumed subsequent increase of the ***mas*** receptor protein may contribute to anatomical and physiological plasticity that is associated with intense activation of hippocampal pathways.

L3 ANSWER 75 OF 76 MEDLINE on STN DUPLICATE 35
ACCESSION NUMBER: 90222168 MEDLINE
DOCUMENT NUMBER: 90222168 PubMed ID: 2109324
TITLE: RTA, a candidate ***G*** ***protein*** - ***coupled*** ***receptor*** : cloning, sequencing, and tissue distribution.
AUTHOR: Ross P C; Figler R A; Corjay M H; Barber C M; Adam N; Harcus D R; Lynch K R
CORPORATE SOURCE: Department of Pharmacology, University of Virginia School of Medicine, Charlottesville 22908.
CONTRACT NUMBER: F32 CA08262 (NCI)
R01 DK37494 (NIDDK)
T32 HL07284 (NHLBI)
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1990 Apr) 87 (8) 3052-6.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M32098; GENBANK-M35297; GENBANK-M35298
ENTRY MONTH: 199005
ENTRY DATE: Entered STN: 19900622
Last Updated on STN: 20000303
Entered Medline: 19900524

AB Genomic and cDNA clones, encoding a protein that is a member of the guanine nucleotide-binding regulatory protein (***G*** ***protein***)- ***coupled*** ***receptor*** superfamily, were isolated by screening rat genomic and thoracic aorta cDNA libraries with an oligonucleotide encoding a highly conserved region of the M1 muscarinic acetylcholine receptor. Sequence analyses of these clones showed that they encode a 343-amino acid protein (named RTA). The RTA gene is single copy, as demonstrated by restriction mapping and Southern blotting of genomic clones and rat genomic DNA. Sequence analysis of the genomic clone further showed that the RTA gene has an intron interrupting the region encoding the amino terminus of the protein. RTA RNA sequences are relatively abundant throughout the gut, vas deferens, uterus, and aorta but are only barely detectable (on Northern blots) in liver, kidney, lung, and salivary gland. In the rat brain, RTA sequences are markedly abundant in the cerebellum. RTA is most closely related to the ***mas*** oncogene (34% identity), which has been suggested to be a forebrain angiotensin receptor. We cannot detect angiotensin binding to the RTA protein after introducing the cognate cDNA or mRNA into COS cells or Xenopus oocytes, respectively, nor can we detect an electrophysiologic response in the oocyte after application of angiotensin peptides. We conclude that RTA is not an angiotensin receptor; to date, we have been unable to identify its ligand.

L3 ANSWER 76 OF 76 MEDLINE on STN DUPLICATE 36
ACCESSION NUMBER: 90136024 MEDLINE
DOCUMENT NUMBER: 90136024 PubMed ID: 2515412
TITLE: Adaptive evolution of ***G*** - ***protein*** ***coupled*** ***receptor*** genes.
AUTHOR: Yokoyama S; Isenberg K E; Wright A F
CORPORATE SOURCE: Department of Ecology, Ethology and Evolution, University of Illinois, Champaign 61820.
CONTRACT NUMBER: MH00635 (NIMH)
SOURCE: MOLECULAR BIOLOGY AND EVOLUTION, (1989 Jul) 6 (4) 342-53.
Journal code: 8501455. ISSN: 0737-4038.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Space Life Sciences
ENTRY MONTH: 199003
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 20000303
Entered Medline: 19900315

AB The phylogeny and patterns of nucleotide substitutions in the visual pigment genes, adrenergic receptor genes, muscarinic receptor genes, and in the human ***mas*** oncogene were studied by comparing their DNA sequences. The evolutionary tree obtained shows that the visual pigment genes and ***mas*** oncogene form one cluster and that the receptor genes form another. In the evolution of rhodopsin genes, synonymous substitutions outnumber nonsynonymous substitutions. This is consistent with the neutral theory of molecular evolution. However, the early evolutionary stages of alpha- and beta-adrenergic and muscarinic receptors are notable for significantly more nonsynonymous substitutions than synonymous substitutions, suggesting the acquisition of novel functiona adaptations. Variable rates of nonsynonymous changes in different domains of these proteins reveal DNA segments that might have been important in their functional adaptations.